THE
LARYNGOSCOPE.

VOL. XLV JANUARY, 1935. No. 1

SYMPOSIUM ON THE VIIIth NERVE.

I.—BASIC PRINCIPLES UNDERLYING TESTS OF HEARING.*

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One attending a course such as we are now having cannot fail to be impressed with the diversity and detail embodied in what we call otolaryngology. To us have been been assigned the problems of deafness. We respectfully suggest that, in routine otolaryngologic practice, there is a vital need for precise diagnostic study of patients with defective hearing. At first thought, some of you may consider that there is no real need for such precise measurement. In fact, in routine practice, precise hearing measurements are the exception rather than the rule. We also recognize this fact. As one of you, just before the meeting, expressed it, "If we do find an old conductive or perceptive lesion, what can be done about it — how can we restore the hearing?" Of course, we quite agree that, in a broad sense, very little can be done to restore the hearing of those who are chronically deaf. This hard fact has always been true, is now, and probably always will be. However, we

*This and the two following papers were lectures before the Midwinter Clinical Course in Ophthalmology and Oto-Laryngology of the Research Study Club, of Los Angeles, January, 1934.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 15, 1934.

do not think that this is the end of the matter, or that we should take the attitude that nothing can be done for the patient. We call to your attention the following benefits for the patient that can be obtained only by precise studies:

1. In the prevention of deafness. Prevention, of course, would be the great solution to the whole problem. As a matter of fact, we all realize that the patients do not come to us, with rare exception, until they have more or less established or fixed lesions and are already appreciably deafened. However, even when such patients have come to us, we have been overlooking the incipient cases — by the very reason of the nature of our old tests. Unless we make precise measurements in a quiet room, we shall keep right on registering a patient as normal, who actually has only 75 or 80 per cent hearing — just as all of us must acknowledge we have been doing throughout all our years of practice. Surely, these are the very patients for whom preventive measures could be undertaken. On the one hand, the otolaryngologist is often discouraged and naturally pessimistic regarding the chronic cases that come to him; on the other hand, we believe that a little contemplation will make all of us realize that in many cases, by early diagnosis, we are able to arrest the progress of the pathologic process in the ear and may look forward to saving such individuals from a condition which means ostracism from many things in life.

2. In precision of diagnosis, in order to guide our treatment. This enables us to decide which patients would benefit from treatment and which would only be wasting their time and money in taking unnecessary treatments.

3. In determining the progress of the deafness, one way or the other, by periodic measurements. Such precise measurements give us a definite concept of the patient's problem and help us to guide him, occupationally and socially.

4. In determining the effect of treatment, either surgical or medical, by charting the patient's hearing acuity, before and after such treatments.

5. In prescribing for certain patients the best type of artificial aid to hearing, to meet the needs of the individual case.

The whole problem might be summarized by asking three questions and answering "yes" to each one:

1. Question: Are there certain conditions of the ear in which the impairment can be relieved and the hearing improved? *Answer:* Yes.

2. Question: Are there certain conditions of the ear in which the impairment cannot be relieved and the hearing cannot be improved? *Answer:* Yes.

3. Question: Is it true, regardless of the nature of the hearing defect, that something can be done to improve and relieve the condition of the individual himself? *Answer:* Yes.

In the course of our three lectures we plan to discuss these problems and to suggest practical methods of handling them. Practical methods of testing and charting hearing will be our problem tonight. The second problem will be that of diagnosis by means of tests and charts, to determine the precise extent of a lesion and to differentiate between that type of deafness due to a conductive lesion, and that type due to a perceptive lesion. The third lecture will deal with the responsibility which the otolaryngologist has in prescribing for his patient, the best artificial aid for his individual condition.

For so many years, we have all recognized the great need for standardization of hearing tests. This problem has received considerable attention during recent years, and has benefited very materially from fundamental researches in acoustics. It is true that we do not as yet have a standardized method of conducting hearing tests; however, we are on the very threshold of the standardization of units and scales for the measurement of hearing. In other fields of science and technology it is difficult to appraise highly enough the value of standardization. For so many years, we have had standard units of length, mass and time; standard units and scales for the measurement, for example, of electrical, thermal and luminous energy; and nearly all the clinical measurements we make are of such a nature that we can express the results in absolute terms which have the same quantitative significance everywhere. The same sort of standards are needed in tests of hearing.

There is at present a national committee in this country which is working on the standardization of units and scales for the measurement of the intensity of sound. This committee is operating under the auspices of the American Standards

Association; it is sponsored by the Acoustical Society of America. United in this movement of standardization are more than 20 physical, technical and medical societies. At the present time we already have units and scales for the measurement of sound intensity, so completely established in English-speaking countries as to make their general adoption a certainty — probably within the next year. It will be only a short time before otologists will be making their measurements in terms of these standardized units and scales. Under these circumstances, we thought it might be of value to you to consider these units at the present time.

The "decibel" throughout all English-speaking countries, has become the standard unit for measuring the intensity of sound. The "decibel" is abbreviated "db." The term "bel"

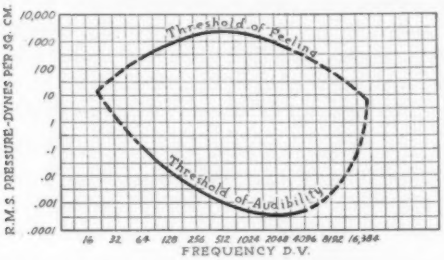


Fig. 1. Curves showing the threshold of audibility and the threshold of feeling for the normal.

was chosen in honor of Alexander Graham Bell. For most measurements the "bel" is a rather large unit, and consequently a smaller unit, the decibel, has come into general use. More and more, the German scientists are coming to use a unit of the same value; as yet they prefer to give the unit the name "phon." This same unit, with the name decibel, will probably become adopted universally, as the standard unit for measuring sound intensity. It is already generally regarded as the rational unit to adopt for the measurement of hearing function. For this reason, it will not be long before otologists will become familiar with this unit; and then they will express the results of their hearing tests in terms of the decibel. For the sake of simplicity, however, particularly for the benefit of the patient, we prefer to express the results of hearing

tests in "percentage hearing," which, nevertheless, is based directly upon the decibel scale.

Suppose we take a sound intensity which is just barely audible to the average individual with normal hearing. This is called the zero level of sound intensity. The value of this zero level is given in the lower curve in Fig. 1.[1] This lower curve represents the intensity which is just barely audible to the average person with normal hearing, for pure tones of different pitch. In this curve the results are expressed in terms of the pressure variations in the ear canal, measured in root mean square dynes per square centimeter. It will be observed that the zero level of sound is dependent upon the pitch of the sound. Specifically, the sensitivity of the ear is greatest for frequencies between about 1000 and 4000 cycles. The sensitivity of the ear becomes less and less, for tones above or below this middle range.

The decibel unit is logarithmic. It corresponds fairly closely with the sensation of the sound, especially for tones above about 700 cycles per second. A sound which is just barely audible has a level of zero decibels. If the amplitude of vibration of this sound (which is just barely audible) be increased tenfold, it will have a level of 20 decibels. An increase in the amplitude of vibration of tenfold corresponds to an increase in the intensity of one hundredfold. The decibel difference between two tones of the same pitch having intensities I_1 and I_2 is ten times the logarithm to the base ten of I_1/I_2, that is,

$$\text{Difference of level in db.} = 10 \log_{10} I_1/I_2,$$

where I_1 is the intensity of the louder tone.

The upper curve in Fig. 1 corresponds to intensities which are so loud that they are felt as well as heard, and it thus might be termed the upper limit of hearing. Since, at this intensity, the sound is felt as well as heard, it is often called the "threshold of feeling" curve.

The noise in this auditorium at the present moment is about 35 or 40 db. You note especially the noise from the ventilating fans. The average loudness of speech in a large auditorium is about 50 db. The loudness of the average individual's speech in this room is about 60 db. In certain modern airplanes the noise is only 68 db. The noise at Seventh and Spring streets, in Los Angeles, during hours of normal traffic,

or inside a Pullman car with the windows closed, is about 70 db. The noise near the elevated train in New York, Chicago or Philadelphia is about 80 to 85 db. The roar of a lion, if you are standing nearby, would be about 90 db., and the noise near a riveter may be as high as 100 db. The report of a cannon is surely as high as 130 db., which is at the upper limit; that is, it is painfully loud.

For sounds which are just barely audible in the range of 1000 to 4000 vibrations per second, the pulsations of the sound waves within the external canal are so small that the amplitude of vibration of the drum-membrane is only about one-billionth of an inch. In response to ordinary conversation, the amplitude of vibration of the drum-membrane is about one-millionth of an inch, or about 1,000 times greater than that of the barely audible sound. In response to painfully loud sounds, the drum-membrane has an amplitude of vibration of about one-thousandth of an inch — that is, about 1,000,000 times the amplitude of a barely audible sound. From the barely audible sound to the painfully loud sound, therefore, the amplitude of vibration of the drum-membrane increases about one millionfold. Since the energy of vibration is proportional to the square of the amplitude, the energy or intensity of a painfully loud sound is a million times the intensity of a barely audible sound. This gives us some conception of the wide scope of response of the human ear to sound vibrations.

Each vertical division in Fig. 1 corresponds to an increase in intensity of tenfold; that is, it corresponds to a difference in level of 10 db. Thus, for a frequency of 512, there are 12.8 such units or scale divisions between the curve for the minimal threshold of audibility and the curve for the threshold of feeling. That is, the threshold of feeling is ten times 12.8, or 128 db. above the threshold of audibility.

The intersections of the two curves in Fig. 1 determine very accurately the lower and upper limits of pitch that the human ear can detect. These limits correspond to a frequency of 20 cycles per second at the lower end, and 20,000 cycles per second at the upper end. Of this total range, 20 to 20,000, the human ear hears best those frequencies between 500 and 5000. Curiously enough, this embraces the most important portion of the range of frequencies of the average human

voice. In other words, the human ear is equipped to hear best the range of the human voice. Although of course speculative, it seems highly probable that the human ear was developed first, and that human speech is a much later development. In the process of evolution, when primitive creatures first crawled out of the water, they came into a new environment on the land. They already had the vestibular part of the ear, but not the auditory part. Gradually, from their needs, they developed the cochlea. The animal had been able to detect motion by means of the vestibular end-organ. He now needed to detect motion from the outside world. The motion-detecting organ was there, and after the oval window had opened the way to impulses from the outside world, he could then utilize this further development of this organ for the detecting of motion. He then had the posterior part of the internal ear for the detection of motion within himself, and the anterior part for the detection of (vibratory) motion from without, which we merely happen to call "hearing." The range of hearing was presumably determined by the need to detect the ordinary noises of the outside world. Later, it would seem that the noises which an animal made with its larynx, and also the noise of the human voice, were adapted to the frequency range of the ear which showed the greatest acuity. Whether or not it be true that the ear came first and the voice came later, the fact remains that the human voice utilizes the most sensitive part of the human auditory mechanism. We have presumably developed our speech and later our music, in such a way as to make them best adapted to our sense organ of hearing.

Under average conditions, except for music, we have very little need for acuity of hearing, or any hearing at all, for the uppermost tones. In our clinical experience, we have found only one person who could hear above 20,000 cycles — a young girl whose upper limit reached to 22,000. The average human being with normal hearing does not hear higher than about 18,000 cycles; and even 18,000 is rarely heard by those who have reached middle age. When one attains an age of 35 or 40 years the limit is down to approximately 15,000. When one has attained an age of 55 to 65 years the average normal limit is approximately 10,000 to 12,000; those who have reached an age of 70 or 75 years rarely hear above 8,000 cycles. There is this sliding scale for the normal, based solely

upon age. We should, therefore, take into consideration the age of the patient before concluding that any observed lowered upper limit of hearing is pathologic.

In the following table, in the column to the left, are given the frequencies of vibration for the tones usually used in testing hearing. In the column to the right are given the number of decibels between the minimal threshold curve and the curve for the threshold of feeling. This chart, which is obtained directly from the curves in Fig. 1, represents the findings for the average normal.

TABLE I.

Frequency of tone in cycles per second	Number of db. between the threshold of hearing and the threshold of feeling
64	63
128	91
256	113
512	128
1024	132
2048	128
4096	116
8192	91

Referring to a frequency of 512, the person with normal hearing will just barely hear a 512 tone, when the tone produces a pressure variation within the ear canal equal to one-thousandth of a dyne.

It will naturally require a greater pressure than this to produce a barely audible tone for a person who has a hearing impairment. Thus, if a pressure of one-hundredth dyne is required for a tone of 512 cycles, the person shows a hearing loss of 20 db. for this tone. Similarly, if a pressure of one-tenth dyne is required, the individual would show a hearing loss of 40 db.; and if a pressure variation of one dyne were required to produce a barely audible sound at 512, the individual would show a hearing loss of 60 db.

In Fig. 2 are plotted the results of a hearing test on the right ear of a certain patient. This chart indicates a hearing loss of only 2 db. at 64 cycles, a hearing loss of 19 db. at 512 cycles, and a hearing loss of 67 db. at 8192 cycles. The shaded area between the two curves represents the frequencies and intensities — the so-called hearing area — which can

be heard by an individual having the type of impairment represented by the hearing loss curve in Fig. 2. The ratio of this shaded area to the entire area between the total loss of hearing curve and the normal hearing curve represents the fractional part of hearing which this patient possesses. Thus, if the shaded area were equal to the unshaded area, the patient would have 50 per cent of hearing. For all purposes of diagnosis, and for the prescribing of artificial aids to hearing, it is always better to compute the percentage of hearing at each frequency. This can be done by means of a hearing curve, such as is shown in Fig. 2, and the data given in Table I. Thus, according to the curve in Fig. 2, there is a hearing loss of 19 db. at 512 cycles. This individual, therefore, has a range of only 128 minus 19, or 109 db. between the minimal thresh-

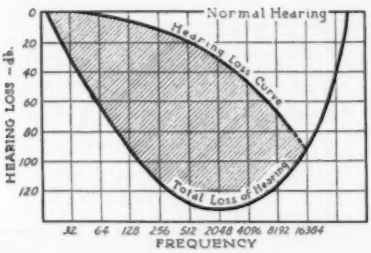


Fig. 2. Charts used by the Western Electric Co. for charting the hearing loss of a patient.

old and the threshold of feeling, whereas the individual with normal hearing has the full range of 128 db. The percentage hearing at 512 cycles for the individual whose hearing is charted in Fig. 2 is rated as 109/128 x 100, or 84 per cent. The curve in Fig. 3 shows the percentage hearing for the patient whose hearing loss in db. is plotted in Fig. 2.

The charts shown in Figs. 2 and 3 represent the commonly used methods of charting and hearing. Fig. 2 represents the method used by the Western Electric Co., and Fig. 3 represents the method which we use.

We consider that the percentage hearing curves, such as the one shown in Fig. 3, is the most satisfactory way of charting the results, at least from the standpoint of the clinician. It seems to us that it is so much simpler, especially for us as

clinicians, to use the "percentage hearing," rather than the "loss in decibels." Incidentally, it helps us to show the patient, in a graphic way, that everything above the curve represents the percentage he does not hear, and everything below the curve represents what he does hear. Otologists, as well as patients, are more accustomed to think in terms of "percentage hearing," rather than in "decibels of hearing loss."

However, even to those of us who know very little about recent developments in acoustics, it is a simple matter to convert from the one system to the other by means of the data given in Table I. Thus, if "HL" represents the "hearing loss" in decibels at any one frequency, the percentage hearing at this frequency is equal to the number of decibels between the two threshold curves, as given in Table I, minus "HL,"

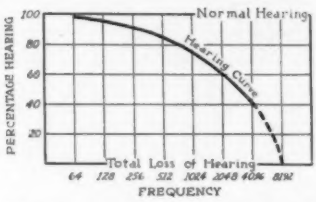


Fig. 3. Charts used by the writer, for charting the hearing loss of a patient.

all divided by the total range, as given in Table I, and all multiplied by 100. Expressed in equation form, the "percentage hearing," abbreviated "PH,"

$$PH = \frac{DR - HL}{DR} \times 100,$$

where "DR" gives the total "decibel range" between the two threshold curves, as specified in Table I.

NECESSITY FOR QUIET.

Tests should not be made in the presence of any noise. It is as necessary to have a quiet room for making reliable hearing tests as it is to have a dark room for conducting fundamental tests of vision. Noise of any kind in a room naturally produces a masking effect on the hearing of tones, especially interfering with the detection of minor gradations near the lower threshold of audibility, so important in hearing tests.

The amount of noise present in the average otologist's office in a metropolitan location is rarely less than 25 to 30 db. In many offices the noise is at a level as high as 40 or even 50 db. If one listens to the tones produced by tuning forks in such rooms, the tones from the forks do not become inaudible at the true minimal threshold of audibility; they become inaudible at the threshold of the noise present in the room — that is, when the tone can no longer be heard *because of the noise.* Unless, therefore, the patient cannot hear the noise in the room, what one measures is not the amount of hearing that the patient has, but rather the amount of noise which is present in the room. This gives very little information concerning the amount of hearing of the patient. It nullifies the whole test. If the hearing measurements are made in a room in which there is noise, we are not giving the patient an

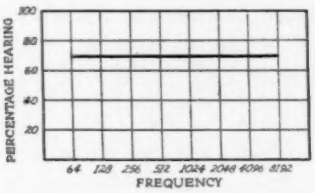


Fig. 4. Chart representing two entirely different individuals: (1) A normal person tested in a noise which produces a deafening effect of 30 per cent, and (2) a patient with a hearing loss of 30 per cent.

accurate measurement of his hearing, unless, of course, his deafness has progressed so far that he cannot hear the noise in the room. To be sure, you might ask the patient, "Can you hear the noise in the room?" If he cannot hear any noise, then it is possible to make an accurate test. But if, on the other hand, he can hear any noise in the room, the test will be inaccurate just to the extent of the amount of noise which he can hear. This is certainly a serious indictment of the customary methods of testing the hearing, which we have been using all these years. In brief, it means that in the past, we have been determining the amount of noise in our rooms, rather than the hearing acuity of our patients. This exhibits the futility of attempting to study our patients unless the tests are made in the quiet.

In Fig. 4, the average hearing acuity is shown as 70 per cent. This same audiogram may represent two entirely dif-

ferent situations. First, it may represent a patient with a hearing loss of 30 per cent. But it may also represent any individual with perfectly normal hearing, being examined in a noisy office which produces a deafening effect of 30 per cent at all frequencies. The chart would be the same for either the deafened patient or the normal. A patient with this amount of deafness is just as well off, for the hearing of speech or music, in this noisy room, as is a normal individual. Such a patient can hear only 70 per cent, because of his hearing defect. An individual with normal hearing can also hear only 70 per cent, because of the masking effect of the noise in the room. According to our old and customary standards of comparing the patient with ourselves (as arbitrary normals), this 70 per cent of hearing acuity would be recorded as 100 per cent. If the tests are made in a noisier office, or in a street car, automobile or railroad train, a similar deafening effect is produced, but even to a more marked degree. In an environment of 60 db. of noise, a patient with a hearing loss of 60 db. hears as well as those of us who have normal hearing. On the one hand, the patient does not hear any sounds below this level, because his hearing is impaired by his lesion; on the other hand, we normals do not hear other sounds below this level, because our hearing is disturbed by the noise; that is, the noise masks all useful sounds which are less than 60 db. above the minimal threshold.

Applying this to tuning forks, our 512 fork can be heard by a person with normal hearing for 58 seconds after it has been given a standard excitation in a soundproof room, such as in the Acoustical Laboratory at the University of California at Los Angeles. If this same fork be given a standard excitation in the average otologist's office, the tone of the fork will not remain audible more than about 30 to 35 seconds; that is, the fork becomes inaudible when the tone has decayed to the level of the noise in the room. If, therefore, the noise level in a room is 30 db. for a tone of 512 cycles (and there are few offices which are quieter than this), a tone such as may be produced by a 512 fork will not be heard unless it is more than 30 db. above the minimal threshold of audibility, owing to the masking effect of the noise. If a person has a hearing loss of 30 db. for 512 cycles, he will just be able to hear the noise in such a room, but he will hear the tuning fork as long as will a person with normal hearing. The hear-

ing tests, as conducted in this room, would therefore fail to detect any impairment of hearing, even though this patient really had a hearing loss of 30 db., which at this frequency is 77 per cent of normal hearing. In brief, a patient with 77 per cent of normal hearing comes to you and you record that he has normal hearing. Consequently, all patients who are tested in such a typical office, by means of tuning forks, the monochord, or the ticking watch, will receive a hearing ratio of 100 per cent, unless they have less than 77 per cent of normal hearing. In a very noisy office, such as are common in the big cities, the noise may be so loud that a person with as little as 60 per cent of normal hearing may be recorded as having normal hearing. It is surely imperative, therefore, that hearing tests, especially those made with a tuning fork, be made in a quiet room. The reliability of any hearing test, on anybody, anywhere, will depend upon the degree of quiet in the room.

In providing such quiet rooms, there is a very reasonable and practical objection that has been raised — the expense. The cost of providing a soundproof room for hearing tests is prohibitive to most of us. If we should pay as much as one or two thousand dollars in the attempt to make a room soundproof, even then the result may not be satisfactory. Noises are transmitted through the floor, ceiling and the walls. In large office buildings the noises produced by the elevators and other equipment are transmitted through the steel frame or masonry partitions. However, this problem can be solved in a practical way. It is very simple to provide a "booth" or "cabinet" which is sufficiently soundproof to exclude the noises encountered in the moderately quiet office. A fairly satisfactory booth, which can be erected for approximately one hundred dollars, has been described by us elsewhere.[2] Any engineer with a knowledge of architectural acoustics can design similar or more elaborate booths.[3]

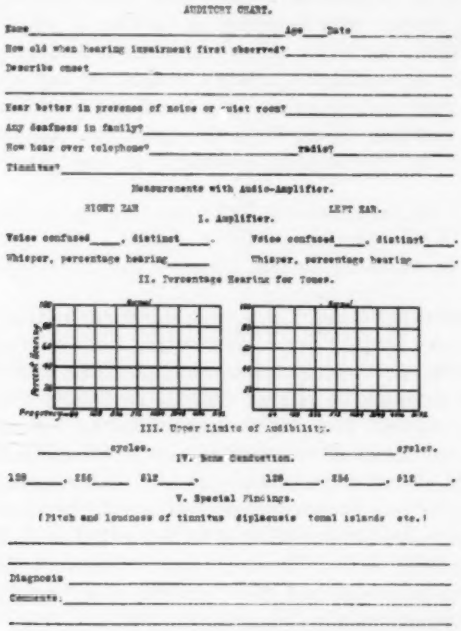
With the use of such a soundproof booth, one is able to detect the early beginnings of a hearing defect. This puts us in a position to cope with the problems of prevention. Our greatest usefulness lies in detecting the very beginning of a hearing defect, early enough to be able to do something about it. Naturally, the ideal would be to give periodic hearing tests to all individuals. This, of course, will probably never happen

— although very many people do have their eyes examined, simply to find out if they might perhaps have a visual defect. One thing we can do. If all of us should make routine tests — if only for the low, medium and high tones — on all our patients who do not come to us complaining of any hearing defects, but for symptoms referable to the nose or throat, it is highly probable that we would find that many of them would show an appreciable loss of hearing, even though they had always considered that they had perfect hearing. Anyone, in the average walks of life, may have no more than 80 or 85 per cent of hearing and yet never suspect that he has any hearing defect whatever. Is it not just this type of case in which we can give the greatest help, by instituting measures to prevent deafness?

HEARING TESTS WITH TUNING FORKS.

In the quiet, it is possible to make reliable hearing tests with a set of tuning forks. In most cases it will be sufficient to use only three forks — 128, 512 and 2048 forks — corresponding to low, medium and high pitched tones. It is necessary, however, that these forks be suitably calibrated and that a standard method of striking the forks be used for exciting them into vibration. The fork must be calibrated with respect to: *1.* its initial intensity, and *2.* its rate of decay. This can be done either by the manufacturer of the fork or at any reputable acoustical laboratory. It is necessary to know the intensity, in db., of each fork immediately after it has received a standard blow. If the fork be allowed to fall from a vertical position through an arc of 90 degrees, hitting a suitable pad (such as soft rubber or felt for the low pitched forks, and hard rubber for the high pitched forks), with a little practice the initial intensity can be reproduced to an accuracy of one or two db. In some of our work, we use a spring hammer, which provides a standard blow for striking the forks. It is then necessary to know the rate of decay of the fork. In general, the rate of decay, in decibels per second, will be a constant. That is, the amplitude of vibration dies away logarithmically. This simply means that the intensity of the tone, expressed in decibels, dies away at a uniform rate. The 512 steel fork, in general, will have an initial intensity — when held in front of the ear canal — of about 70

to 80 db., and it will die away at a rate of about 1.2 to 1.7 db. per second. Lower pitched forks decay more slowly, and higher pitched forks decay more rapidly. The 2048 fork, at least, should be a massive steel fork or one of the Duratone type, with a low damping rate, and therefore dies away more slowly than does an ordinary steel fork. If the initial intensities of the forks be known (when the forks are held


AUDITORY CHART.

Name_____ Age___ Date_____

How old when hearing impairment first observed?_____

Describe onset_____

Hear better in presence of noise or quiet room?_____

Any deafness in family?_____

How hear over telephone?_____ radio?_____

Tinnitus?_____

Measurements with Audio-Amplifier.

RIGHT EAR LEFT EAR.

I. Amplifier.

Voice confused____, distinct____. Voice confused____, distinct____.

Whisper, percentage hearing_____ Whisper, percentage hearing_____.

II. Percentage Hearing for Tones.

Normal

Normal

Percent Hearing

III. Upper Limits of Audibility.

_____cycles. _____cycles.

IV. Bone Conduction.

128____, 256____ 512____. 128____, 256____, 512____.

V. Special Findings.

(Pitch and loudness of tinnitus diplacusis tonal islands etc.)

_____

_____

Diagnosis _____

Comments._____

_____

Fig. 5. Auditory charts used by the writer.

immediately in front of the ear canal) and the rates of decay of the forks be known, it is then possible to make precise hearing tests in the quiet, and to express the results in standard or absolute units. Thus, suppose the 512 fork produces an initial intensity of 70 db. and that it dies away at the rate of 1.5 db. per second; suppose further that when this work is used for testing the hearing acuity of a certain patient it is found to remain audible for 20 seconds. In 20 seconds the

fork would have diminished in intensity from 70 db. to 40 db.; that is, the patient would just barely hear a tone of 512 cycles which had a level of 40 db. Hence, this individual would have a hearing loss of 40 db. or (referring to Table I) 69 per cent of normal hearing. Similar tests made with the 128 and 2048 forks, or with any other forks which are calibrated, would determine the hearing loss or the percentage hearing at these frequencies for air-conduction. The results could then be plotted in decibel loss as in Fig. 2, or, as we prefer it, in per cent hearing, as in Fig. 3, and the plotted results would represent an air-conduction audiogram for the patient. It is possible to prepare charts for any set of tuning forks, so that one measures only the duration of audibility of the fork, and then reads from the chart the "hearing loss" in decibels or in "percentage hearing," at each test frequency.

A chart for three tuning forks used by the writers is given in Table II. By the use of these charts in the quiet, reliable tests can be made by tuning forks — for air-conduction.

For bone-conduction it is a very different matter. A tuning fork is a very limited device for testing bone-conduction. The tuning fork is not designed to communicate a large amount of energy through its shank. This is especially true for high frequency forks. Thus, it is impossible to obtain sufficient energy from forks above 2000 cycles to be audible by bone-conduction, even for individuals with normal hearing. A chart similar to the one shown in Table II should be used for bone-conduction tests, but it will have to be limited to forks of lower pitch than 2000 cycles. These charts can be prepared for other sets of tuning forks, and the "hearing loss," or "percentage hearing," read directly from the charts.

A serious limitation in the use of tuning forks should be mentioned. Since the initial intensities of tuning forks are far below the threshold of feeling, they are useless for testing the hearing of those who have gross impairments. For example, with the forks of Table II, it would be impossible to measure the hearing of those who have less than 34 per cent hearing at 128 cycles, less than 42 per cent at 512 cycles, or less than 51 per cent at 2048 cycles. Such patients would not hear the forks, and consequently they would be given the same rating as those who are totally deaf.

TABLE II.

Chart for Converting Duration of Audibility of Tuning Forks Into "Hearing Loss" (in Db.) and "Percentage Hearing." (For Air-Conduction.)

	128 fork (steel)		512 fork (steel)		2048 fork (Duratone)	
Initial intensity in db.	58		72		62	
Rate of decay in db./sc.	1.1		1.5		3.7	
Duration of Audibility	Hearing Loss Db.	Percentage Hearing	Hearing Loss Db.	Percentage Hearing	Hearing Loss Db.	Percentage Hearing
50 secs.	3	97				
45 secs.	8	91	5	96		
40 secs.	14	85	12	91		
35 secs.	20	78	20	84		
30 secs.	25	72	27	79		
25 secs.	31	66	35	73		
20 secs.	36	60	42	67		
15 secs.	42	54	50	61	7	95
10 secs.	47	48	57	55	25	80
5 secs.	53	42	65	49	44	66
2 secs.	56	38	69	46	55	57

BONE-CONDUCTION.

As just mentioned, a set of calibrated tuning forks can also be used to a certain extent for making precise measurements of hearing acuity by bone-conduction. As in the case of air-conduction tests, the results should be stated in terms of decibels "loss of hearing" or "percentage hearing." The loss of hearing by bone-conduction is determined — in the case of the tuning fork tests — by measuring the duration of audibility of each tuning fork when the shank of the fork is pressed against the mastoid bone, in the usual manner. If the patient can hear any noise in the room, the bone-conduction tests, just as the air-conduction tests, will be inaccurate. The measurements will again determine the amount of masking produced by the noise in the room rather than the hearing of the patient by bone-conduction. We have always been accustomed to speak of bone-conduction as equal to normal, less than normal, or greater than normal. Bone-conduction has been recorded as "increased" or "decreased." We shall show in the next lecture that "increased" bone-conduction is rarely found among those with even gross conductive lesions. The routine tests of bone-conduction which all of us have been making in the past have indicated that persons with conduc-

tive impairment have so-called "increased" bone-conduction solely because the person with conductive impairment does not hear the noise in the room, whereas the person with normal hearing experiences a hearing loss of 20 to 30 db., owing to the masking effect of the noise. Bone-conduction tests which are conducted in a noisy room, therefore, do not give reliable measurements unless the patient is entirely deaf to the noise; and then the tuning forks or other devices must have been calibrated in the quiet. The use of a soundproof booth is thus seen to be a prime necessity for making all bone-conduction tests, whether with tuning forks or with audiometers. In brief, absolute quiet is highly desirable in testing the air-conduction. It is absolutely necessary in testing bone-conduction.

HEARING TESTS WITH VACUUM TUBE DEVICES.

In recent years many devices employing vacuum tube circuits have been developed for making hearing tests. These devices are usually called audiometers, and are very convenient and reliable for making hearing tests. For one thing, the test room does not need to be absolutely quiet for the air-conduction tests, since the ear is covered with a telephone receiver which, when held tightly against the ear, will exclude most of the noise encountered in a reasonably quiet room. Under present conditions, in the noisy rooms that are in common use, these vacuum tube devices are far superior to tuning forks. However, even with these vacuum tube instruments, it is essential that the bone-conduction tests be conducted in the quiet. Audiometers are calibrated by the manufacturer, so that they read directly either in "hearing loss" in decibels, as in the Western Electric audiometer, or in "percentage hearing," as in our audio-amplifier. In these devices, pure alternating currents are generated by a vacuum tube oscillator and a telephone receiver. An attenuator — a volume control — is placed between the oscillator and the telephone receiver so that the loudness of the tone can be varied by known gradations, either in decibels or in percentage hearing. The bone-conduction receiver may consist of a special type of "strip" diaphragm or a moving coil to which is attached a metal rod about four to six inches long, the distal end of which is pressed against the mastoid process.

Fig. 1. The door leading into the soundproof room—closed. The edges of this door are lined with rubber so that the door fits tight.

Fig. 2. The door is opened, and the door of the soundproof booth can be seen inside.

Fig. 3. Test with the amplified voice and with the amplified whisper, inside the soundproof booth.

Fig. 4. Test with pure tones, to determine the minimal threshold of hearing, throughout the range of hearing.

In making hearing tests with audiometers, the hearing acuity should be determined at different frequencies both by air-conduction and by bone-conduction, and the results plotted in charts, as in Fig. 2 or Fig. 3. (At this point was demonstrated the technique of making measurements with the Western Electric audiometer and with our audio-amplifier, followed by an examination of a patient with the audio-amplifier.)

Fig. 5. Test for pure tones, with a noise apparatus in opposite ear.

Fig. 6. Test for the minimal threshold of audibility, by bone conduction. The bone conduction can be tested with the audio-amplifier, from the lowest frequencies up to 4096.

Fig. 5 shows the chart which we use for recording the results of hearing tests. The chart is for the most part self-explanatory. The first part of the chart gives the history and a description of the onset, together with other information which is useful in classifying the type of deafness. The next part of the chart gives the results of voice and whisper tests conducted with the amplifier part of the instrument. The patient listens to the amplified voice of the examiner and answers whether the voice seems distinct or confused. The whisper test is conducted by whispering at a standard intensity into the microphone and controlling the intensity with

Fig. 7. The audio-amplifier shown in the open room, in order that the technique may be more clear. Test of the amplified voice and the amplified whisper.

Fig. 8. Test for the minimal thréshold of audibility, throughout the range of hearing.

the attenuator in the telephone receiver circuit. On the next part of the chart are recorded the audiograms for the right and left ears both by air-conduction and by bone-conduction. The use of such charts in determining the type and extent of deafness will be discussed fully in the following paper. In the next part of the chart are recorded the upper limits of hearing. Part IV is for the results of the bone-conduction tests. Any problem of special interest, such as the characteristics of tinnitus, diplacusis or tonal islands, are recorded in

Fig. 9. Test for the minimal threshold of audibility, with noise apparatus in opposite ear.

Part V. The remainder of the chart is used for recording the diagnosis and any special comments with regard to the case.

In conclusion, it should be mentioned that tests for hearing by means of the speaking voice, the ticking watch, the whisper or tuning forks which are moved away from the ear until inaudible, are only qualitative, and should be regarded as inadequate for the routine tests of hearing. In particular, the whisper and the ticking watch can test only the acuity for high pitched sounds. The voice and whisper tests are subject to considerable error, depending upon the initial intensity of

the spoken or whispered word; again, the intensity of these tones depends upon the acoustical properties of the entire room in which the tests are made. Finally, the results obtained by such tests ordinarily cannot be expressed in terms of absolute units, such as "db. hearing loss" or "percentage hearing." In brief, hearing tests should be made only in a quiet room, either by calibrated tuning forks, or by one of the audiometric methods. Reliable tests can be made by means of the tuning forks if the forks are properly calibrated, and if the examiner has acquired the proper technique of making the tests. It is much easier and it takes so much less time to make hearing tests by means of audiometers, and the results in general will be more reliable than those obtained with tuning forks. However, results obtained with the tuning fork in the quiet and by a trained technician are likely to be more reliable than tests made by an untrained technician with the most elaborate audiometer, when such tests are made in a noisy room. Hearing tests should always be made by a trained technician, and if we are going to discover those who have slight hearing impairments, for whom there is some hope of treatment, the tests must be made in the quiet.

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3. See, for example, Chaps. IX, X, XI, XII and XIII, "Architectural Acoustics," by V. O. Knudsen (John Wiley and Sons, 1932), which treat the subject of sound insulation in considerable detail.

SYMPOSIUM ON THE VIIIth NERVE.

II.—DIAGNOSIS OF HEARING IMPAIRMENTS.

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Los Angeles.

In general otolaryngologic practice, a carefully taken history is of such vital importance that a clue to the proper diagnosis and treatment is often furnished merely by the story of the patient's experiences. The diagnosis of deafness, however, does not come under this general rule. The history of the patient, even though it includes the detailed story of all of the patient's experiences concerning his hearing, does not, as a rule, give us much help in diagnosis. Occasionally the history is helpful. For example, if the patient states that he began to notice a hearing impairment at about 18 years of age; that the hearing gradually became less and less acute; that he seems to hear better in the presence of a noise; and that members of his family have noticed a similar experience, in beginning to become hard-of-hearing at an early age, with out any apparent cause, — naturally the diagnosis of otosclerosis is suggested. Again, if the patient states that he hears better in a quiet place, and that he is very much confused and is unable to distinguish conversation when many people are speaking in the same room — we naturally think that the patient is suffering from a cochlear lesion. Similarly, if the patient states that he has a tinnitus that sounds like the beating of his pulse, we think it probable that the middle ear is involved; if he complains of a tinnitus that sounds like escaping steam, we think of a cochlear defect. However, in the diagnosis of deafness, the history of the patient usually provides only a general, and often hazy concept of the nature of his lession.

The actual inspection of the ear, nose and throat frequently enables us to make a diagnosis of the cause of deafness. However, without precise measurement of the function of hearing, we are unable to have a complete knowledge of the nature, as well as the degree, of the hearing impairment. One is frequently surprised to find that the functional tests

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 15, 1934.

entirely change the concept that was furnished by the history and by the inspection of the ear, nose and throat.

There are these three factors, helpful in making the diagnosis: *1.* The history of the patient; *2.* the direct inspection of those parts of the ear, nose and throat that can be seen; and *3.* the determination of the condition of those parts that cannot be seen. In routine practice we find that it is useful, first to take the history; then to make the hearing tests and the vestibular tests; and then, with the knowledge of the condition of those structures that cannot actually be seen, finally to make the usual direct inspection of the ear, nose and throat.

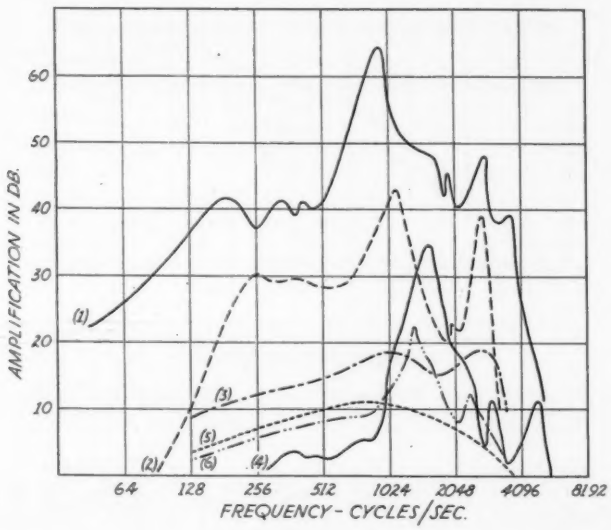
The diagnosis of hearing impairments is, therefore, largely based upon the determination of certain physical characteristics of hearing. If tests similar to those described in the preceding paper have been carefully made, it is possible to determine these physical characteristics, and from them to ascertain with unfailing accuracy both the type and the degree of any hearing impairment. Unfortunately, it is not possible always to make a precise diagnosis as to the condition, or conditions, that have produced the lesion. A study of any individual will not always determine the cause, but it does give us the type and the degree of the defect. The present paper will discuss the use of hearing tests and hearing charts in making a diagnosis. Routine tests which are based upon the use of the ticking watch, the spoken or whispered voice, the monochord, or the tuning fork (as commonly used) are capable of detecting gross impairments of hearing and, in most cases, of determining in general whether the impairment is of the conductive or the perceptive type, but they are incapable of determining the proportion of conductive and perceptive impairment in cases of mixed deafness, and they are most apt to fail in detecting an impairment in its early stages — that is, at the very time when detection may be the means of suggesting treatment which may arrest the further progress of the deafness.

In the following, we shall assume that the tests of hearing are conducted in a quiet room, and that the technique of making the tests is in conformity with the principles set forth in the preceding paper. The cases we shall discuss were studied by our audio-amplifier at the office, in a soundproof

booth. Most of the cases were also tested in the soundproof room at the University of California at Los Angeles. Of course, the same results could have been obtained by means of any good audiometer, or even by a set of calibrated tuning forks, provided that the technique was good and that the tests were made in a sufficiently quiet room. As we have already emphasized, the degree of quiet required depends upon the degree of impairment of the patient tested. It is necessary, in any case, that the room be quiet enough so that the patient does not at any time hear the noise in the room.

The differential diagnosis between conductive and perceptive impairments is naturally based upon measurements of the auditory acuity, both by air-conduction and bone-conduction. In general, air-conduction tests offer a means for determining the *over-all* characteristics of the organ of hearing, including both the conductive and the perceptive mechanisms. On the other hand, bone-conduction tests enable us to make a fairly direct test of the cochlea, since the sound vibrations reach the cochlea through a path which is largely independent of the middle ear mechanism. In all routine tests of hearing which are commonly used by otologists, the tests are intended to disclose the patient's acuity by air-conduction and by bone-conduction; and also, to compare the patient's acuity by bone-conduction with that of a normal. Usually the examining otologist assumes that his own hearing, both by air-conduction and bone-conduction, is normal. This assumption is not warranted. It should not be assumed, if accuracy is to be attained. It is customary to assume that if both air-conduction and bone-conduction are less than normal, the impairment is essentially of the perceptive type. It would seem that this conclusion is justified. On the other hand, it is generally stated that if air-conduction is less than bone-conduction, and the bone-conduction is "greater than normal," the impairment is of the conductive type. There is, however, an element of uncertainty and of fallacy in this last test, which assumes that conductive impairment is characterized by a bone-conduction which is greater than normal. We wish to emphasize, from the practical standpoint, what we have outlined in the previous lecture. The result obtained in the average office, which is not free from noise, depends directly upon the extent of the noise in the room, the degree of impairment of the individual tested, and the hearing acuity of

the so-called "normal" — *i.e.*, the examiner. No one exhibits a normal bone-conduction if there is any noise in the room. The examiner, therefore, is using a false standard for the normal, in the average office. Owing to the presence of the noise in the room, the examiner does not determine his acuity for bone-conduction. He merely determines the loudness of that tone which is just barely masked by the noise in the room. In other words, he has measured the amount of noise in the room rather than his own true bone-conduction. The patient with the conductive impairment does not hear the



noise in the room (he at least does not hear it so loudly as does the normal), and since his cochlea usually has normal acuity, he will exhibit an apparent acuity by bone-conduction which is greater than that for the "normal" in the noisy room. If you will test a patient in your office and find that the bone-conduction is "greater than normal," as we have always considered it to be, and then test him again in a soundproof room, you will find that the "greater than normal" has vanished, and that his bone-conduction is either

normal or less than normal. It is absolutely necessary to make appropriate corrections for all bone-conduction tests, which are made in rooms not free from noise. It is far better and, incidentally, much simpler, to make the tests in a soundproof room. It is then not necessary to apply any correction, and the results obtained in different rooms and by different examiners will be not only comparable, but accurate.

A suitable and modern slogan for the otologist might be, "bigger and better bone-conduction." We have always studied such a long frequency range by air-conduction and such a short range by bone-conduction. We have studied air-conduc-

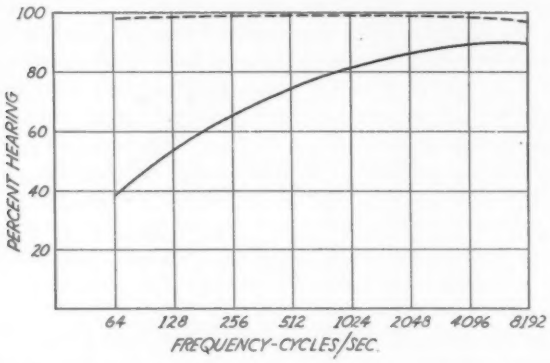


Fig. 6. Typical chart of a patient with a purely conductive impairment. The solid line curve gives the per cent hearing by air conduction, and the dotted line gives the hearing by bone conduction.

tion, as it were, from Los Angeles to New York, but bone-conduction only from Los Angeles to Pasadena. Bone-conduction tests over a wide range of frequencies give a much better knowledge of the condition of the cochlea than we can obtain from air-conduction tests — in fact, they furnish almost a direct test of the cochlea, whereas air-conduction tests furnish only a knowledge of the composite condition of the middle and internal ear mechanisms. In this sense, thorough bone-conduction tests are analogous to the study of the fundus of the eye by the ophthalmoscope. It has always been impossible for us to study bone-conduction, except in a very narrow band of frequencies, simply because tuning forks above 2000 cannot be heard by bone-conduction. We feel that many otolo-

gists do not realize the advantage of having a really good bone-conduction test, to help them in diagnosis. Often an audiogram is made, for air-conduction, throughout the entire frequency range, whereas the bone-conduction is determined only for a single frequency. We are coming to realize that a diagnosis becomes much more simple and accurate, if we determine both the air-conduction and the bone-conduction throughout a wide range. In most cases it is necessary to test only three frequencies by bone-conduction — the low, medium and high; for example, 128, 512 and 2048 cycles per second. With a good audiometer and a suitable bone-conduc-

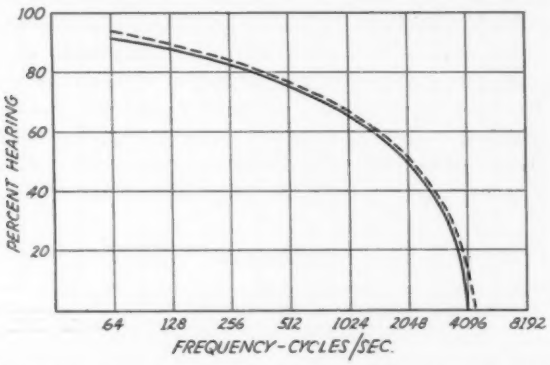


Fig. 7. Typical chart of a patient with a purely perceptive impairment. Note that the air conduction and bone conduction curves are identical.

tion attachment, bone-conduction tests can now readily be made up to 4096 cycles. Summarizing, if we limit our bone-conduction tests to only a tuning fork or two, in the usual low frequency register, we are making a wide range air-conduction test and a very short range bone-conduction test. If we do this, we are denying ourselves the valuable diagnostic information that can be obtained from the wide range bone-conduction tests.

If the patient is markedly deaf in only one ear, or if there is a considerable difference in the acuity of the two ears, it is necessary to take precautions to avoid the conduction of sound through the head, from one ear to the other. The insulation through the head amounts to approximately 50 db. in

the air-conduction tests. It is of the order of only 3 to 5 db. in the bone-conduction tests. When the sound-waves enter through the air, there is very little energy transmitted through the skull. In bone-conduction, however, the entire skull is set into vibration. Therefore, in testing cases of monolateral deafness, especially in bone-conduction tests, it is necessary to use some sort of noise apparatus which will mask the hearing of the better ear, while the poorer one is being tested. An ordinary electric buzzer connected to a telephone receiver, such as is used in our audio-amplifier, is serviceable for this purpose. Any noise apparatus, however,

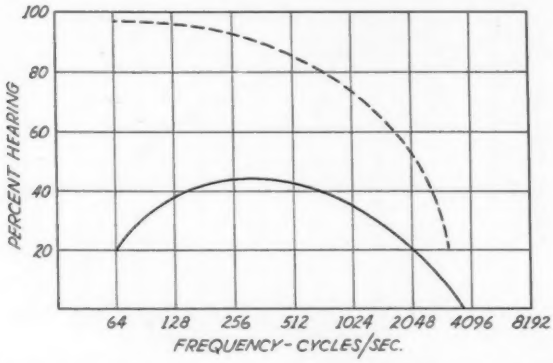


Fig. 8. Typical chart of a patient with mixed conductive and perceptive impairment.

must not be too loud, because the excessive noise would naturally mask the hearing, in the opposite ear which is being tested.

Figs. 6, 7 and 8, respectively, give typical audiograms, both for air-conduction and bone-conduction, of a pure conductive impairment, a pure perceptive impairment, and a mixed conductive and perceptive impairment. The percentage hearing by air-conduction is indicated by the solid line curve, and the percentage hearing by bone-conduction is indicated by the dotted line curve.

In the purely conductive type (see Fig. 6), the bone-conduction is normal for all tones. This means that the cochlea has normal acuity. The loss of acuity by air-conduction is

greatest for the tones of low pitch, and the acuity increases with frequency, becoming nearly normal for tones of very high pitch.

In the purely perceptive type (see Fig. 7), on the other hand, the curves for air-conduction and bone-conduction are practically identical. This means that all of the defect is within the cochlea; that is, it makes no difference whether the sound reaches the cochlea by the normal path through the middle ear or through the cranial bones, independently of the middle ear. The greatest loss of acuity is for tones of high pitch, and the acuity is relatively good for tones of low pitch.

In the mixed type of deafness (see Fig. 8), the acuity by air-conduction is greatest for tones in the middle register, and the acuity decreases both for lower and for higher tones. The bone-conduction is normal, or nearly normal, for tones of low pitch, but drops off quite markedly for tones of high pitch, approaching the air-conduction curve for tones of very high pitch. These three charts are typical of the three general classifications of hearing defect.

CASE REPORTS.

The following case reports are arranged under the main headings of (1) Conductive, (2) Perceptive, (3) Mixed, and (4) Malingering:

Under the Conductive Cases are given otitis media chronic catarrhal; a case of unusual middle ear lesion, in which the patient's statement as to the type of tinnitus led to the diagnosis; otitis media chronic purulent (two cases); otosclerosis (four cases), and stapes fixation, unilateral due to a staphylococcus septicemia.

Under the Perceptive Cases are given cochlear lesions from chronic toxemia; cochlear lesion, acute, from typhoid fever; Paget's disease, involving the cochlea; neuroma of the VIIIth nerve; pressure on the VIIIth nerve from a carcinoma in the cerebellopontile angle.

Under the Mixed Cases are otosclerosis, later complicated by a cochlear lesion from toxemia; two cases, each having both conductive and perceptive lesions, both apparently due to a chronic tonsillar infection.

Malingering, one case.

CONDUCTIVE CASES.

Mrs. G. L. M.: Note the normal bone-conduction throughout the range, indicating normal cochlear function. The normality of the internal ears is corroborated by the 100 per cent vestibular function. This is the ideal type of a case in which the deafness is arrested and the tinnitus stopped by removing the cause. It is only unfortunate that the tonsillectomy and submucous resection of the septum could not have been done several years before. However, as mentioned before, the patients do not come to the otolaryngologist during the early stages in the

development of the lesion. This patient can hear fairly well if people speak to her with a little more than ordinary loudness. For the theatre, and under special conditions, an artificial aid is helpful.

Mr. S. V.: At times one is led along the right pathway to a diagnosis by some statement of the patient, which may seem of comparatively little importance. This patient had noted an impairment of hearing, particularly in his right ear, but his main anxiety was caused by many sudden attacks of vertigo, which he had had very frequently, over a period of four years. He had been able to continue with his work, but he was living constantly in the fear of one of these sudden attacks. He had been very carefully studied throughout this period of four years, along almost every avenue of approach, from gastrointestinal studies to the concept

AUDITORY CHART.

Name Mrs. George L. M. Age 41 Date

How old when hearing impairment first observed? 32

Describe onset Came on gradually from age 32 to age 36. Then tonsilectomy and resection of Septum.

Hear better in presence of noise or quiet room? Noise.

Any deafness in family? None

How hear over telephone? Well, radio? Well

Tinnitus? Constant, in both ears, for 2 years.

Measurements with Audio-Amplifier.

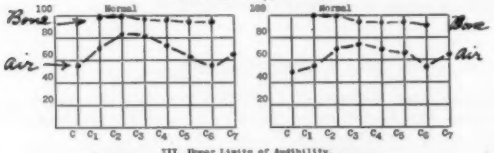
RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused ____, distinct V. Voice, confused ____, distinct V.

Whisper, percentage hearing 80. Whisper, percentage hearing 73

II. Percentage Hearing for Tones.



III. Upper Limits of Audibility.

16,000 d.v. 16,000 d.v.

IV. Bone Conduction, in Office

128 97, 256 103, 512 100. 128 103, 256 109, 512 103

V. Special Findings.

(Pitch and loudness of tinnitus, displacusis, tonal inlands, etc.)

Diagnosis: O.M.C.C.

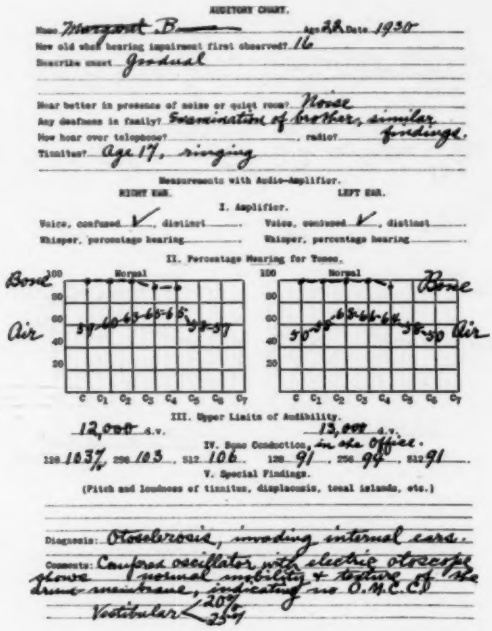
Comments: Vestibular < 100% / 100%

The progressive deafness & tinnitus ceased, after the operations — nine years ago.

of a brain tumor. The statement of the patient, which led to the diagnosis was, "I have almost constantly had a noise in my right ear; there has been no roaring or hissing, but I am always conscious of the beating of my pulse in the right ear. Whenever this sound in my right ear becomes louder, I know that an attack of vertigo is coming on. It has almost invariably happened that when this noise starts to become louder in my right ear, I will have an attack in from 15 to 20 minutes. This increase of the noise in my ear gives me this warning, and in that way I am enabled to prepare for an attack." The type of tinnitus suggested that the lesion was in the right middle ear. The comprex oscillator with electric otoscope showed an impairment of mobility of the drum-membrane and hammer handle. The auditory chart indicated a conductive lesion. Blowing air through a catheter into the right Eustachian tube

produced one of his typical attacks. On two occasions when we did this he stated "that is exactly the sensation that I have when I have one of my attacks." The next day he had an attack while driving his automobile. He shut off the electric switch and then, according to our instructions, he yawned. The attack ceased at once. The septum was deflected so markedly to the right that he had practically no ventilation through the right naris. Since a submucous resection of the septum, 11 months ago, he has had no further attacks.

Mr. R. A. H.: Seen in 1929 at age 28 years. Chronic purulent otitis media, right ear; chronic purulent healed in left middle ear; ethmoiditis, bilateral, anterior and posterior, chronic, with polypoid degeneration;


AUDITORY CHART.

Name Margaret B—— Age 28 Date 1930

How old when hearing impairment first observed? 16

Describe onset Gradual

Hear better in presence of noise or quiet room? Noise

Any deafness in family? Examination of brother, similar findings.

How hear over telephone? , radio?

Tinnitus? Age 17, ringing

Measurements with Audio-Amplifier.

RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused , distinct . Voice, confused , distinct .

Whisper, percentage hearing . Whisper, percentage hearing .

II. Percentage Hearing for Tones.

III. Upper Limits of Audibility.

12,000 c.v. 12,000 c.v.

IV. Bone Conduction, in the Office.

128 16/37 256 105 512 106 128 91 256 94 512 91

V. Special Findings.

(Pitch and loudness of tinnitus, diplacousis, tonal islands, etc.)

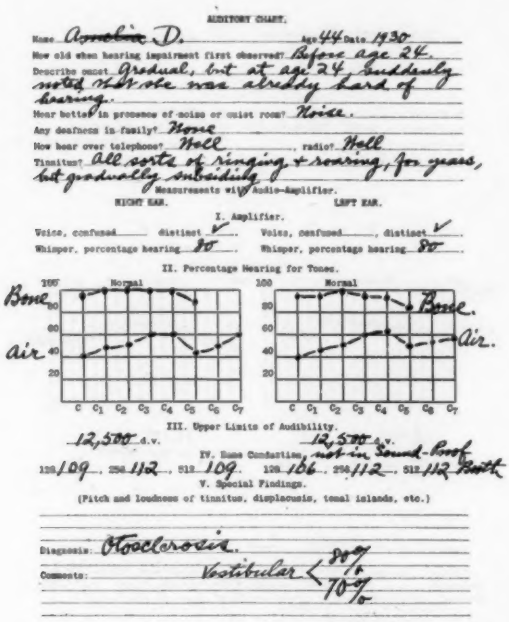
Diagnosis: Otosclerosis, involving internal ears.

Comments: Confirmed oscillator with electric otoscope; drum membrane, indicating no O.M.C.D.

Vestibular < 20/ 25/

irregularly shaped tonsils, very hard and deeply buried, giving the appearance that they had been cut, although they had not; old chronic pharyngitis. Almost continuous discharge from the right ear since age 4 years. Normal cochlear function was demonstrated. The amplified conversational voice was heard distinctly; the upper limit of hearing was normal; the bone-conduction normal. After operation on the tonsils, and later on the sinuses, the right middle ear ceased to discharge and the hearing improved so markedly in both ears that he was able to continue with his occupation, which he felt would soon be lost to him because of his deafness. For the past four years he has been free from discharging ear and his hearing in each ear, which originally averaged 65 per cent, now averages 77 per cent, so that now it is not necessary to recommend an artificial aid to hearing.

Miss A. B.: Seen in 1928. Her ears had been discharging almost continuously "all her life," according to her mother's statement, and she was referred for bilateral radical mastoid operation. Cochleas were perfectly normal. The amplified conversational voice was heard distinctly; the upper limit was normal, and the bone-conduction was normal. The patient was very fortunate in having normal cochleas, because the vestibular function was definitely impaired, suggesting that there had been an invasion of the vestibular function of each ear from the chronic otitis media. The hearing defect was due entirely to a marked bilateral conductive lesion, which was obvious on inspection of the middle ears. Treatment consisted of removal of the adenoid remnants and of small bits of tonsil tissue; the velum palati was brought forward by means


AUDITORY CHART.

Name Annabelle D. Age 44 Date 1930

How old when hearing impairment first observed? Before age 24.

Describe onset Gradual, but at age 24 suddenly noted that whole was already hard of hearing.

Hear better in presence of noise or quiet room? Noise.

Any deafness in family? None

How hear over telephone? Well, radio? Well

Tinnitus? All sorts of ringing + roaring, for years, but gradually subsiding

Measurements with Audio-Amplifier.

RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused ____ distinct ✓. Voice, confused ____, distinct ✓.

Whisper, percentage hearing 80. Whisper, percentage hearing 80.

II. Percentage Hearing for Tones.

III. Upper Limits of Audibility.

12,500 d.v. 12,500 d.v.

IV. Bone Conduction, not in Sound-Proof

128 109, 256 112, 512 109. 128 106, 256 112, 512 112 Both

V. Special Findings.

(Pitch and loudness of tinnitus, diplacousis, tonal islands, etc.)

Diagnosis: Otosclerosis.

Comments: Vestibular < 80% to 70% o

of a catheter through the nose and the adenoid remnants were teased away under direct inspection. Later polyps were removed from both middle ears. The suppuration ceased in both ears and the ears have remained dry for five years. Like the previous case, the patient had feared that she would lose her position because of her deafness. Her hearing acuity was increased an average of 15 per cent and she is earning her living as a telephone operator. It is necessary to see her only six times a year, except occasionally when she has a recurrence of her sinuitis, in which case she reports immediately for treatment.

Miss M. B.: Seen in 1930. The hearing impairment was first noticed at age 16 years, the onset was very gradual, and at age 17 years she began to have ringing noises in both ears. During the past six years she has become more and more deaf, but the noises have become less and

less noticeable. Her brother's deafness came on in exactly the same way and the examination of the brother also led to a diagnosis of otosclerosis. Note on the chart that the amplified voice is definitely confused and that she has a marked vestibular defect. The demonstration by the comprex oscillator, of the normal texture of the drum-membrane was of great help in this case; also the normal bone-conduction. It appears that the otosclerosis has invaded both internal ears, and therefore it was necessary to select an artificial aid for hearing that would not furnish much amplification.

The particular test which confirmed the diagnosis of otosclerosis was with the comprex oscillator and electric otoscope. Each drum-

AUDITORY CHART.

Name Irwin B. Sim Age 49 Date 1912

How old when hearing impairment first observed? 45

Describe onset Gradual, left ear 9 years ago. Also became conscious of marked impairment in right ear during past 2 years.

Hear better in presence of noise or quiet room? Quiet.

Any deafness in family? None

How hear over telephone? Only fairly well. radio? fairly well.

Tinnitus? Buzzing in both ears, first noticed in left ear.

Measurements with Audio-Amplifier.

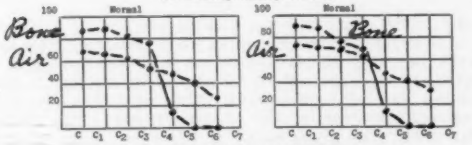
RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused V, distinct ____. Voice, confused ____, distinct fairly

Whisper, percentage hearing O. Whisper, percentage hearing O

II. Percentage Hearing for Tones.



III. Upper Limits of Audibility

8000 d.v. 8,000 d.v. in office.

IV. Bone Conduction.

128 97, 256 91, 512 100. 128 95, 256 91, 512 94.

V. Special Findings.

(Pitch and loudness of tinnitus, diplacusis, tonal islands, etc.)

Diagnosis: Cochlear defect, bilateral.

Comments: Vestibular < 15% 10%

membrane was normally movable in all its parts. The hammer handles also moved normally. The texture of the drum-membrane was normal. This would seem to rule out otitis media chronic catarrhal, as there would certainly be some impairment of mobility, or change in the texture of the drum-membrane, if a middle ear lesion were to account for such a marked conductive defect.

Mrs. C.: Patient gave the following history: Had a severe illness, from which she nearly died, one and a half years ago. Her illness began with a throat infection, followed by a staphlococcus septicemia. The diagnosis of her conductive deafness in the right ear appears to be a bony overgrowth around the right oval window, due to the staphlococcus septicemia. She had a severe osteomyelitis of the spine, and at the same

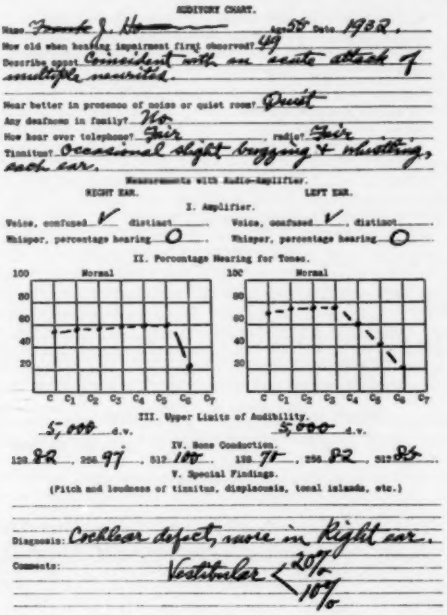
time she became deaf in the right ear. Examination showed the left ear to be practically normal. In the right ear, the air-conduction curve showed no hearing for the lowest tones, a better hearing for the high tones, and an almost normal upper limit — the classical curve of a conductive deafness. Bone-conduction was heard much better in the right ear than in her left (normal) ear. Immediately the question arose as to what sort of a lesion this could be in order to produce such a marked deafness, and yet be of the middle ear, conductive, type. The Weber was markedly referred to the right—the deaf ear. In the right ear, the air-conduction was much less than the bone-conduction—in fact, the bone-conduction was normal. The diagnosis appeared to be very definite, and yet it was so unusual that naturally one had reservations in stating such an opinion. The comprex oscillator with electric otoscope, demonstrated normal texture and mobility of both drum-membranes — in other words, there was nothing in the middle ear itself that could account for this conductive impairment. The only possible place where this lesion could be is around the oval window; therefore, we had a condition similar to otosclerosis, entirely confined to the right ear; as she was 38 years old, by that time surely, if it were otosclerosis, we would expect that the left ear would have been at least somewhat involved. The patient was assured that she did not have otosclerosis, and that the lesion had happened once and for all, during her staphylococcus septicemia. The patient was particularly relieved to be told that there was not any reason why she should have any trouble whatever in her left ear. The status, as here presented, is just the same today as it was three years ago.

PERCEPTIVE CASES.

Mr. I. B. S.: This chart gives a classic picture of a defect that is entirely due to a degenerative change in each cochlea. The lower tones are heard well, but the higher tones are scarcely heard at all. The downward curve, as we approach the higher frequencies, is shown, both for air-conduction and bone-conduction. Note also that the chart of the right ear is almost identical to that of the left ear. The history is that of a perceptive defect; he hears better in the quiet; only fairly well over the telephone; the amplified conversational voice is not heard well; the amplified whisper is not heard at all; the upper limit of hearing is lowered, and the bone-conduction curve corresponds to the air-conduction curve — a perfect picture of a purely perceptive lesion. It is surprising that the hearing defect, and presumably the lesion, is the same in both ears, because, so far as the patient was concerned, he seemed to hear very well with the right ear until only two years ago. There has evidently been a long-standing degenerative change in the left cochlea which matches exactly the more recent change in the right cochlea. This is a typical case of degenerative change in either the cochleas or VIIIth nerves, due to toxins brought there by the blood stream. Note also that the vestibular function is even more impaired than the cochlear. Naturally, it is very important to know whether this degenerative change has been one that has extended over a long period, and apparently the vestibular tests settle this point. As he has had no attacks of vertigo, or other vestibular symptoms, the vestibular degeneration must have occurred over a long period, because, if there had been a sudden insult he surely would have been disturbed with vestibular symptoms. The rather rapid loss of hearing in the right ear, beginning about 20 months ago, suggests there is some focus of infection which still remains. Otosclerosis would seem to be definitely eliminated. The hearing is too good for the very low tones; he does not "seem" to hear any better in the presence of a noise; has no record of any deafness in the family; and his hearing defect was not noticed in the slightest until age 37 years; he states that until he was 37 years of age he heard perfectly in both ears. All of these facts seem to rule out hereditary deafness.

Recommendations: (1) To attempt to find a possible focus of infection or constitutional disease. He is advised to have an exploratory opening made in the canine fossa on the left side and if the mucosa within the maxillary antrum is thickened, to remove it by a submucous resection. (2) A suitable artificial aid for hearing.

The tests were repeated for several artificial aids for hearing, both by the audio-amplifier and by the speech articulation tests, as described in detail in the following lecture. The results show that the best artificial aid actually did improve the percentage of his vowel articulation and consonant articulation. The consonant articulation, however, was improved only 11 per cent by the instrument which the tests proved was


AUDITORY CHART.

Name Frank J. H——— Age 58 Date 1922.

How old when hearing impairment first observed? 49

Describe onset Coincident with an acute attack of multiple neuritis.

Hear better in presence of noise or quiet room? Quiet

Any deafness in family? No.

How hear over telephone? Fair radio? Fair

Tinnitus? Occasional slight hissing & whistling, each ear.

Measurements with Audio-Amplifier.

RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused V distinct ___. Voice, confused V, distinct ___.

Whisper, percentage hearing O. Whisper, percentage hearing O.

II. Percentage Hearing for Tones.

100 Normal 100 Normal

80 80

60 60

40 40

20 20

C C_1 C_2 C_3 C_4 C_5 C_6 C_7 C C_1 C_2 C_3 C_4 C_5 C_6 C_7

III. Upper Limits of Audibility.

5,000 c.v. 5,000 c.v.

IV. Bone Conduction.

128 82 256 97 512 100 128 71 256 82 512 85

V. Special Findings.

(Pitch and loudness of tinnitus, diplacousis, tonal islands, etc.)

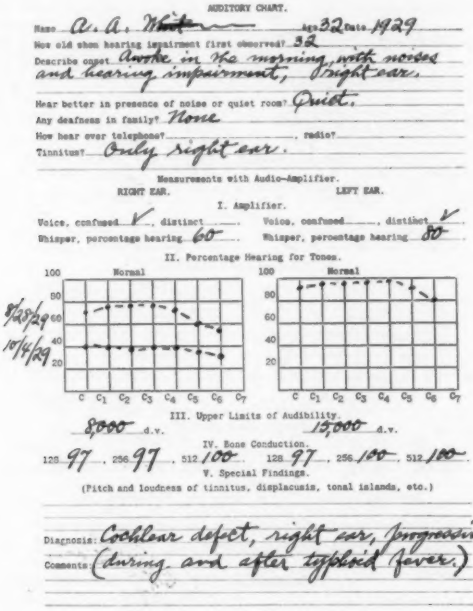
Diagnosis: Cochlear defect, more in Right ear.

Comments: Vestibular < 20% 10%

best for him. Naturally this is all that can be expected of an artificial aid in this case, because amplification soon reaches the upper painful limit because of his cochlear defect. The patient was warned to keep the instrument at "medium," because, if he used full amplification and were to "bombard" his cochlea with too loud a sound, it would be injurious to the already weakened cochlea.

F. J. H.: This case illustrates how little an individual can know about his own hearing defect until precise measurements are made. He had always considered that he could not hear in the left ear, but that he could hear fairly well in the right ear. For years he had made it a point to place on his right side individuals with whom he wished to converse. Examination showed that he actualy heard better in the ear

that he thought was deaf. The chart shows that the hearing is better in the left ear than in the right ear. One of the reasons that led him to consider he was deaf in the left ear was that when he tested himself, he could hear the watch tick in his right ear, but could not hear it in the left ear. Dr. B. Alexander Randall always began his lecture on the functional tests of hearing by saying: "I hear the watch tick very well in my bad ear, but I cannot hear it at all in my good ear." Naturally, it is all a question of the impairment at different frequencies. The average hearing of this patient is only 58 per cent in the right ear, whereas in the left ear it is 70 per cent. The lowered percentage of hearing for the upper frequencies in the left ear makes it difficult for him to hear



AUDITORY CHART.

Name A. A. White Age 32 Date 1929

How old when hearing impairment first observed? 32

Describe onset Awoke in the morning, with noises and hearing impairment, right ear.

Hear better in presence of noise or quiet room? Quiet.

Any deafness in family? None

How hear over telephone? ____, radio? ____

Tinnitus? Only right ear.

Measurements with Audio-Amplifier.

RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused V, distinct ____. Voice, confused ____, distinct V.

Whisper, percentage hearing 60. Whisper, percentage hearing 80.

II. Percentage Hearing for Tones.

9/28/29

10/4/29

100 Normal 90 80 60 40 20 C C1 C2 C3 C4 C5 C6 C7

100 Normal 90 60 40 20 C C1 C2 C3 C4 C5 C6 C7

III. Upper Limits of Audibility.

8,000 d.v. 15,000 d.v.

IV. Bone Conduction.

128 97, 256 97, 512 100 128 97, 256 100, 512 100

V. Special Findings.

(Pitch and loudness of tinnitus, displacusis, tonal islands, etc.)

Diagnosis: Cochlear defect, right ear, progressive

Comments: (during and after typhoid fever.)

the consonants like "th," "s," and "f." Naturally, this does interfere with a proper understanding of ordinary speech. His bilateral cochlear defect was caused by a neuritis, which he had in 1926—a neuritis not only of the VIIIth nerve, but of other nerves. The general neuritis disappeared after his tonsils were removed in 1926. The diagnosis is corroborated by the vestibular tests, which show an average of 20 per cent function right ear and 10 per cent left ear. When the neuritis occurred in both VIIIth nerves, at the same time that the other nerves were being involved, the vestibular and cochlear function of both VIIIth nerves were affected and of course it is very fortunate that it was the vestibular part that was almost destroyed, whereas the auditory portion was only impaired. The vestibular defect is, of course, relatively unimportant in his life and he was simply warned not to go on high places in which

he would have to balance himself, because of the lack of normal motion-sensing impulses from his ears.

A. A. W.: This patient showed, directly under our observation, a progressive vestibular and auditory loss of function, due to typhoid fever, which we did not know that he had when we first saw him. When first seen he showed a very slight cochlear impairment in the right ear, as shown on the chart. Turning him to the right gave a nystagmus of full normal duration, whereas turning to the left gave almost no nystagmus at all, demonstrating a recent vestibular impairment in the right ear. The caloric test corroborated this in that there was no vestibular function in right ear; after four minutes of douching there were no responses of nystagmus, motion-sensing, past-pointing, falling, nausea, pallor or sweat. The very next day the report came back that the Widal reaction was positive. When he recovered from the typhoid fever the hearing was practically gone in the right ear, as noted on the chart. First examination showed normal nystagmus after turning to the right, and none after turning to left; three months later the nystagmus was 21 seconds after turning to the right and 10 seconds after turning to the left; eight months after the original onset the nystagmus was 15 seconds after turning to the right and 14 seconds after turning to the left.

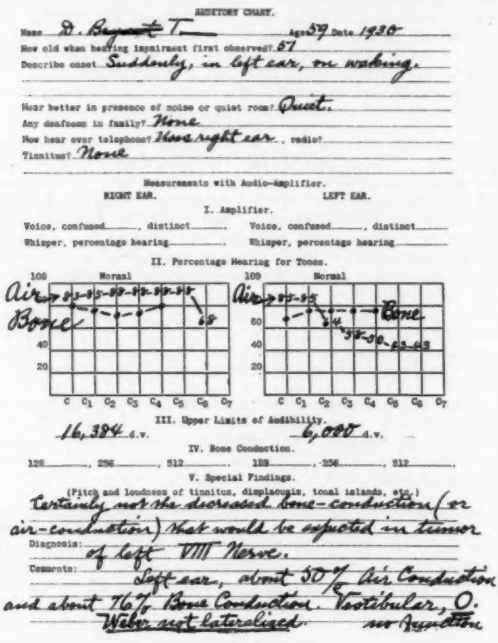
It is not often that we see a case like this. On the first examination we were at a loss to understand the cause of the obvious new involvement of the right internal ear in both cochlear and vestibular portions, only to find out that he had at the moment "walking typhoid fever." Incidentally, such an involvement is not usual in typhoid fever. It is also interesting that after eight months had passed the nystagmus after turning had become equalized so that the responses after turning to the right were just the same as after turning to the left. The right vestibular function was originally destroyed and then the central nervous system established compensation. This compensation, in this case, was evidently complete after a period of eight months. Evidently it requires only eight months, or less, for the central nervous system to bring about equalization. The cochlear lesion will no doubt be permanent.

J. S. B.: Two years before this examination, for no obvious cause, he began to have attacks of vertigo. These attacks have continued from time to time during the past two years. Shortly after the first attacks he rather suddenly lost his hearing in the left ear. This deafness was accompanied by tinnitus, also in the left ear.

Examination of the left ear showed a marked cochlear defect and only 20 per cent vestibular function. At first, it seemed that there was either a neuroma of the left VIIIth nerve or an occlusion of the arterial circulation. The idea of such vascular occlusion, however, was not maintained because the patient has continued to have attacks of vertigo from time to time, month after month. The idea of a neuroma of the VIIIth nerve did not seem probable because there were no intracranial symptoms or findings by Dr. Lyster from the eye standpoint, or Dr. Ingham from the neurologic standpoint. X-rays were taken as a matter of routine to determine whether a neuroma of the left VIIIth nerve had widened the opening of the internal auditory meatus. It had not. But the X-rays revealed the typical mottled appearance of the bones of the head with rarefaction in some areas and an increase of density in other areas, characteristic of Paget's disease. The X-rays of the pelvic bones showed the same condition. It, therefore, seems that we were looking too far afield to explain the invasion of his internal ear if we were to consider anything except Paget's disease, which we do know that he has. The same bony overgrowth could very reasonably have extended into the left cochlea, causing the cochlear defect and the process might be active at the present time, invading the vestibular part of the left internal ear, thereby causing attacks of unsteadiness and vertigo, which he is having

from time to time. In brief, a clinical diagnosis was made of Paget's disease of the internal ear. Incidentally, the patient was much interested in the scientific aspects of his case and he willed his ears to us for histologic examination.

D. B. T.: This patient showed a rather good cochlear function in spite of a neuroma of the nerve itself. Naturally this was disturbing in attempting to make the diagnosis. However, other factors made it possible to make the diagnosis. In December, 1930, when we first examined him, we found that psychic factors were unquestionably responsible for many of his symptoms. However, as Dr. Harvey Cushing has always emphasized, an important and determining factor in angle tumors is a


AUDITORY CHART.

Name D. Bryant T— Age 57 Date 1930

How old when hearing impairment first observed? 57

Describe onset Suddenly, in left ear, on waking.

Hear better in presence of noise or quiet room? Quiet.

Any deafness in family? None

How hear over telephone? Uses right ear, radio?

Tinnitus? None

Measurements with Audio-Amplifier.

RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused, distinct. Voice, confused, distinct.

Whisper, percentage hearing. Whisper, percentage hearing.

II. Percentage Hearing for Tones.

100 Normal 100 Normal

Air 95-95-95-95-95-95-95 Air 95-95 Bone

Bone 60 40 20 C C1 C2 C3 C4 C5 C6 C7

III. Upper Limits of Audibility.

16,384 d.v. 6,000 d.v.

IV. Bone Conduction.

128, 256, 512. 128, 256, 512.

V. Special Findings.

(Pitch and loudness of tinnitus, diplacusis, tonal islands, etc.)

Certainly not the decreased bone-conduction (or air-conduction) that would be expected in tumor of left VIII Nerve.

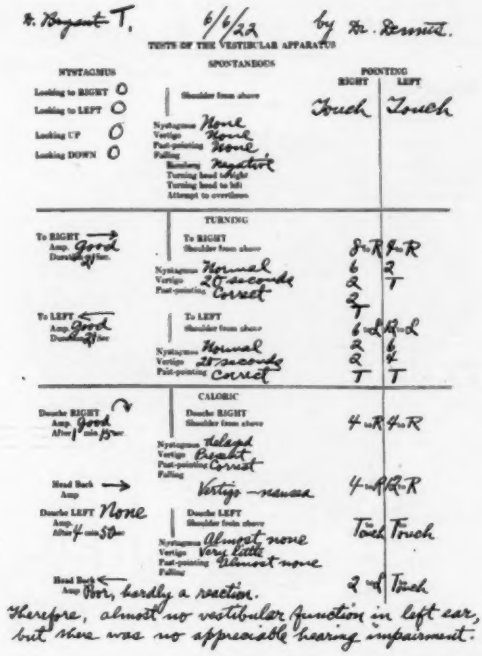
Diagnosis:

Comments: Left ear, about 50% Air Conduction and about 76% Bone Conduction. Vestibular, O. Weber not lateralized. no Rinne.

careful and detailed history. This man's history was definitely that of an angle tumor. He began to grow deaf in the left ear without apparent cause and also began to have tinnitus in March, 1922. Three months later (see accompanying chart) he was examined by Frank L. Dennis, of Colorado Springs, who found almost the identical vestibular picture which we now find, eight and one-half years later. To begin with, therefore, it seemed reasonable to us to conclude that a neuroma began to grow on the left VIIIth nerve, and that was what was the matter with the patient at the time of Dr. Dennis' first and only examination. It also seems that this patient's tumor has ceased to grow, or that it has grown so slowly that intracranial adjustments have been made to accom-

modate it. This latter concept would explain how there could be an angle tumor, even with entire absence of any pressure symptoms or findings. The impaired constitutional responses to the vestibular tests were suggestive of posterior fossa lesion. The diagnosis of angle tumor was finally made, but during the preliminary studies there were two findings that made it difficult to consider that he did have such a neuroma:

(1) The vertical canals of the opposite side produced good responses. Of course, this is now explained in that there evidently was no pressure exerted against the brain-stem at the time of the examination.



Therefore, almost no vestibular function in left ear, but there was no appreciable hearing impairment.

(2) His bone-conduction was excellent in this left ear. Here we have a large neuroma of the VIIIth nerve itself, and yet the auditory fibers were very little involved. He was distinctly uncertain as to the lateralization in the Weber test. He said he heard it about equally in the two ears, and again that he heard it better in the right ear. On repeated tests it seemed that he did hear better in the right ear, but he also did hear it in the left ear, and the final impression was that he heard it only a little better in the right ear. Of course, if the left auditory nerve was destroyed there would be no uncertainty whatever, and he would have promptly stated that he heard it only in the right ear. The air-conduction was no better than the bone-conduction in the left ear, and the bone-conduction in the left ear was good. In brief, although there was no vestibular function in the left ear, the bone-conduction was

almost as good in the left ear as in the right ear. The tests were repeated in the sound-proof room at the University of California at Los Angeles. The air-conduction was good for the low tones in the left ear, but dropped off rather suddenly. This explains why he suddenly woke up one morning, 15 years ago, and "could not hear in the left ear." The impairment had evidently involved the upper range necessary for the hearing of the consonants.

Resume: Deafness and tinnitus in left ear, March, 1922. Vertigo in fall of 1928. In January, 1929, very uncertain in his walking. At this time dull headaches, but no acute pain. Headache began again in May,

1930, and slowly became worse; in June so bad that he had a day and night nurse; much vertigo and staggering; had the nurse for two months. Intermittent headaches; every once in a while, vertigo. September, vertigo almost every day; in past few weeks would have fallen a number of times if someone had not been there to catch him.

Comment: (1) The tinnitus was the first symptom.

(2) Here was a neuroma of the VIIIth nerve and yet the auditory function was not markedly impaired.

(3) The two vestibular examinations, eight and one-half years apart, showed the same findings, although the tumor had no doubt grown very much during that period. This brings in a thought of caution to us all—one can have a good response from the vertical canals of the opposite

side and yet there may be a large angle tumor present. In other words, the vertical canals' fibers from the opposite ear do not seem to be involved except when there is pressure against the brain-stem.

(4) If only we could be more certain in our early diagnosis it would be not only possible, but probable, that these cases could be saved by the early removal of the tumor. Dr. Dennis saw him only once; he exhibited only the vestibular defect and tinnitus; his hearing was normal. If only the patient had continued under Dr. Dennis' care it is probable that the diagnosis could have been made and the tumor removed.

The patient was examined by Dr. Ray G. Taylor: "Stereo radiographs in the Towne position show no definite evidence of erosion of the petrous ridge. The two sides are not entirely symmetrical however; the left side is somewhat flatter and lacking in certain prominences seen on the right ridge."

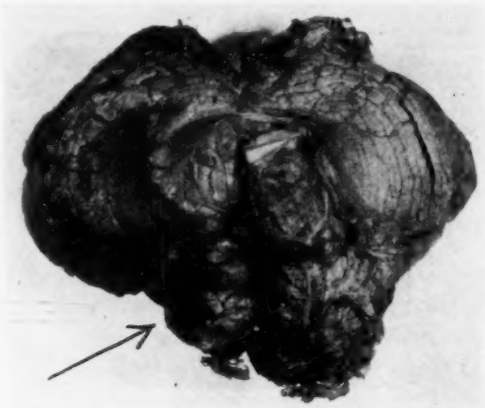


Fig. 10. Brain tumor of the left cerebellopontile angle. Note how the tumor has pressed the brain-stem markedly to the right.

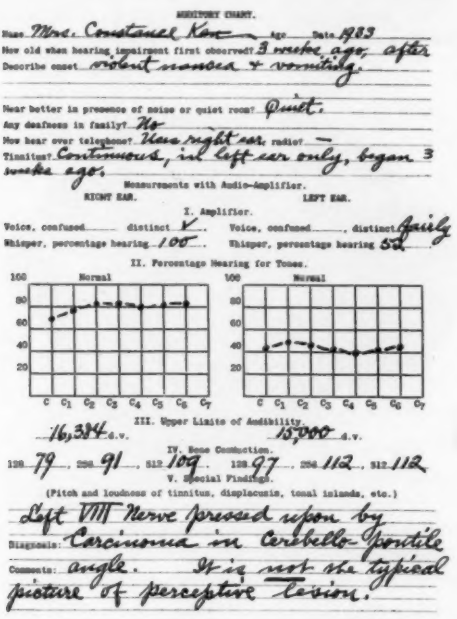
Dr. Cyril B. Courville made the following report: Firm nodular tumor, which measures 3.5x3.7 cm.; yellowish-gray. Tumor compresses the side of the pons and distorts the brain-stem, IVth ventricle and vermis to the opposite side of the midline. Diagnosis: neurofibroma of the VIIIth nerve.

Mrs. C. K.: Carcinoma, metastatic, on the left side of the pons near the foramen magnum.

Right breast removed two and a half years ago; carcinoma. Was well for two years, then came headache, numbness of rectum and vulva, loss of voice, uncertain gait, vertigo, nausea and vomiting, double vision, pain on left side of neck and left ear.

Examination: Dr. Lyster found bilateral choked discs left side, more marked and paresis of left VIth nerve. There was a paresis of the left velum palati, indicating involvement of the left IXth and Xth. Further involvement of the Xth (recurrent laryngeal branch) was demonstrated by paresis of the left vocal cord.

Auditory: The history of sudden deafness in the left ear would cause one to suspect that there occurred a marked involvement of the left VIIIth nerve only three weeks ago, after a violent attack of vertigo, nausea and vomiting. A tinnitus appeared at the same time, left ear, and has continued to date. It is surprising to find that she hears fairly distinctly, with the amplified voice, in this left ear; the upper limit is almost perfect, and the bone-conduction is excellent from the lowest tones up to 512; in fact, for these tones she has perfect bone-conduction. This is a very peculiar finding and might lead us to think that there had occurred pressure against the VIIIth nerve rather than a tumor of the VIIIth nerve itself. An angle tumor growing on the VIIIth nerve of

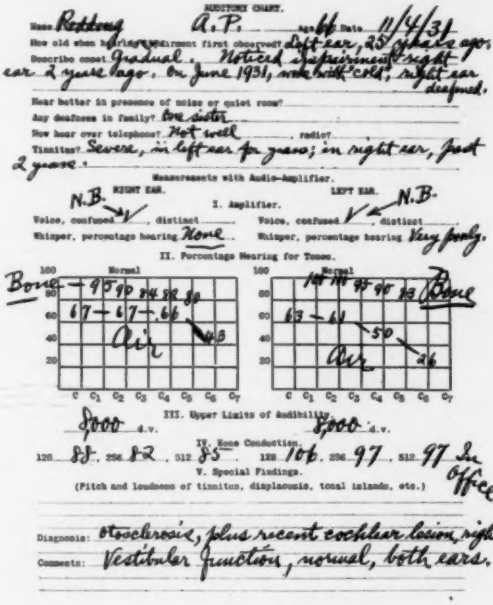

AUDITORY CHART.
Name Mrs. Constance Kon Age Date 1923
How old when hearing impairment first observed? 3 weeks ago, after
Describe onset violent nausea + vomiting.
Hear better in presence of noise or quiet room? Quiet.
Any deafness in family? No
How hear over telephone? Uses right ear, radio? —
Tinnitus? Continuous, in left ear only, began 3 weeks ago.
Measurements with Audio-Amplifier.
RIGHT EAR. LEFT EAR.
I. Amplifier.
Voice, confused distinct V. Voice, confused distinct fairly
Whisper, percentage hearing 100. Whisper, percentage hearing 52
II. Percentage Hearing for Tones.
100 Normal
80
60
40
20
C C1 C2 C3 C4 C5 C6 C7
III. Upper Limits of Audibility
16,384 d.v. 15,000 d.v.
IV. Bone Conduction.
128 79 256 91 512 109 128 97 256 112 512 112
V. Special Findings.
(Pitch and loudness of tinnitus, diplacusis, tonal islands, etc.)
Diagnosis: Left VIII Nerve pressed upon by Carcinoma in Cerebello-pontile
Comments: angle. It is not the typical picture of perceptive lesion.

course would be suspected, because of this deafness in the left ear and the poor responses on the caloric test of the left ear to the caloric test; however, there were so many other findings, such as the paralysis of the left vocal cord, that came on before this trouble in her left ear; a neuroma of the left VIIIth nerve would probably have shown deafness and tinnitus, and possibly staggering, many, many months ago, if it were a neuroma of the VIIIth nerve itself; and finally, the auditory chart, as mentioned above, seems to suggest that the VIIIth nerve has become pressed upon by something outside of itself.

Vestibular: Spontaneous—on looking straight ahead a slight spontaneous nystagmus to the left (seen only with the ophthalmoscope); looking up an oblique nystagmus, up and to the right; on looking down rotary nystagmus to the left. Caloric—right: Prompt rotary nystagmus to the

left, but as the douching proceeded, a large horizontal element entered into it; motion-sensing definitely present, but no pallor, sweat or nausea, even on prolonged duration. Caloric—left: Nystagmus came on promptly after douching, but then disappeared and then reappeared again. Motion-sensing was present, but no pallor, sweat or nausea—just as in the right ear. In brief, the left vertical canals showed very little function and the horizontal canal showed about 25 per cent function.

Comment: The first involvement apparently was of the left Xth. We know that the paralysis of the left vocal cord signifies a lesion at, or below, the left Xth nucleus in the brain-stem. This led us to suspect that the lesion began there. However, if it were a tumor of the brain-stem


AUDITORY CHART.

Name Rettberg A.P. Age 44 Date 11/4/31

How old when hearing impairment first observed? Left ear, 25 years ago; right ear 2 years ago. On June 1931, woke with cold; right ear deafened.

Describe onset: Gradual. Noticed impairment right

Hear better in presence of noise or quiet room?

Any deafness in family? One sister

How hear over telephone? Not well, radio?

Tinnitus? Severe, in left ear for years; in right ear, just 2 years.

Measurements with Audio-Amplifier.

RIGHT EAR. N.B. LEFT EAR. N.B.

I. Amplifier.

Voice, confused V, distinct. Voice, confused V, distinct.

Whisper, percentage hearing None. Whisper, percentage hearing Very poorly.

II. Percentage Hearing for Tones.

Normal 100 80 60 40 20 C C1 C2 C3 C4 C5 C6 C7

Bone — 95 90 84 52 50

67 — 67 — 66

Air 48

Normal 100 80 60 40 20 C C1 C2 C3 C4 C5 C6 C7

Bone 10 10 95 90 83

63 — 61 — 50 — 26

Air

III. Upper Limits of Audibility

8000 d.v. 8,000 d.v.

IV. Bone Conduction.

128 85 256 82 512 85 128 106 256 97 512 97 In Office.

V. Special Findings.

(Pitch and loudness of tinnitus, diplacousis, tonal islands, etc.)

Diagnosis: Otosclerosis, plus recent cochlear lesion, right

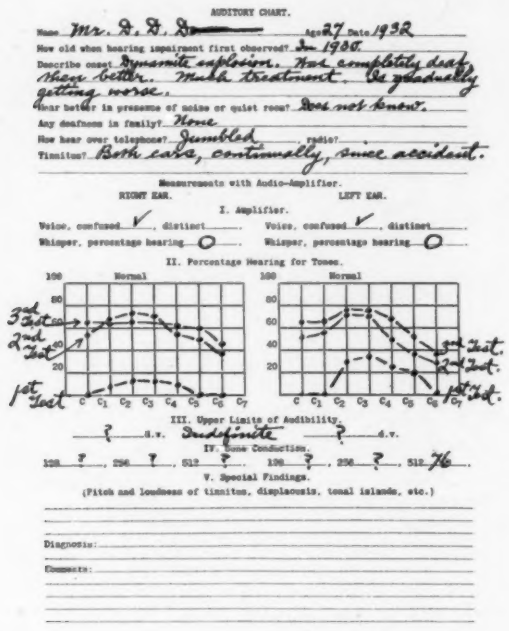
Comments: Vestibular function, normal, both ears.

itself, extending out into the left angle, it would seem probable that she would have definite paralysis of the extremities, either on the same or the opposite side, depending on its relation to the decussation of the pyramidal tract. If it were a tumor of the left VIIIth nerve, involving the other nerves in the angle, one would surely expect she would have had VIIIth nerve symptoms sooner; one month ago she did not have any such symptoms. The symptoms of involvement of other nerves came first. The diagnosis suggested is "that the brain-stem is affected by pressure on the left side involving the various nerves on the left side, as detailed above."

Operation by Dr. Mark Albert Glaser revealed a tumor mass of hard consistency in the left cerebellopontile angle, alongside of the pons, extending from the lower portion of the pons down to the foramen magnum. Histologic report, carcinoma.

MIXED.

A. P. R.: At age 31 years, the hearing in the left ear began to fade away and gradually grow worse until he thought that he had lost all of his hearing in this ear. The hearing in the right ear has been slightly impaired for the past five or six years, but was fairly good for ordinary purposes; then on June 5, 1930, he awoke in the morning with a very violent sore throat and he became deaf in the right ear; no pain; no discharge from ear; great roaring in right ear. The noise was so loud and varied that he could give no definite description of the type of sound. In spite of this history, the diagnosis appears to be definitely otosclerosis, although the history certainly is not characteristic.


AUDITORY CHART.

Name Mr. A. P. R. Age 27 Date 1932

How old when hearing impairment first observed? In 1930

Describe onset Dynamite explosion. Was completely deaf then better. Much treatment. Is gradually getting worse.

Hear better in presence of noise or quiet room? Does not know.

Any deafness in family? None

How hear over telephone? Jumbled radio?

Tinnitus? Both ears, continually, since accident.

Measurements with Audio-Amplifier.

RIGHT EAR. LEFT EAR.

I. Amplifier.

Voice, confused ✓, distinct . Voice, confused ✓, distinct .

Whisper, percentage hearing O. Whisper, percentage hearing O.

II. Percentage Hearing for Tones.

III. Upper Limits of Audibility.

? d.v. Indefinite ? d.v.

IV. Bone Conduction.

128 ?, 256 ?, 512 ?. 128 ?, 256 ?, 512 76.

V. Special Findings.

(Pitch and loudness of tinnitus, diplacousis, tonal islands, etc.)

Diagnosis:

Comments:

Examination with the comprex oscillator and electric otoscope: Right ear—limitation of motion of the hammer-handle, but it is by no means rigid enough to account for his conductive impairment. Left ear—similar, and it is obvious that neither the drum-membrane nor the hammer-handle is responsible for the conductive impairment. Therefore, there is evidently a stapes fixation in both ears.

It would seem that both ears had already been affected by the otosclerosis, but that the threshold of hearing was not sufficiently low for him to notice any impairment in the right ear, and then on June 5, he had an entirely different lesion—a toxic invasion of the right cochlea, which was added to the already existing conductive defect, causing him to become deaf in the right ear.

The air-conduction curve would not make a diagnosis one way or the other; the bone-conduction, however, is so excellent throughout the range that it is very evident that his great defect is conductive. However, there is a cochlear defect in both ears, especially in the left ear, as indicated not only by the history, but by the confused voice sounds when he was spoken to with the audio-amplifier. We could have been led astray in this diagnosis had it not been for the bone-conduction study. An artificial aid for hearing was recommended. He was told that it was unwise for us to attempt to mask his tinnitus with an objective noise (which is very simply made from a radio loud speaker, attaching it directly to the electric light socket), because all objective noises are very disturbing to this man because of his cochlear impairment. Recommended an amplifier for his telephone, which can be arranged with the local telephone company, to be used in the left ear.

Mrs. L. H. H.: 1932. Two years ago, the patient began to have attacks of vertigo, nausea and vomiting; these attacks have continued off and on to date of examination. Five years ago she noted a hearing defect in the left ear.

Examination showed an involvement of both internal ears in both cochlear and vestibular portions; slight cochlear defect in right ear and a marked cochlear defect in the left ear. Vestibular function 40 per cent right ear and 10 per cent left ear. Just before she would have an attack of vertigo the patient noted a clanging noise, as if someone were knocking on a piece of iron; this noise was only in the left ear. After tonsillectomy the patient ceased to have any attacks of vertigo, and repeated examinations of the last two years demonstrate that there has been no further progress in the hearing defect. Apparently this is a clear-cut case of involvement of both internal ears in both cochlear and vestibular portions due to a chronic tonsillitis.

Mr. G. H.: 1932. This is a similar case, except that the deafness was noticed first and the attacks of vertigo, nausea and vomiting came on later. Examination showed a bilateral cochlear defect, an average hearing of 60 per cent right ear and 38 per cent left ear. The vestibular function, 10 per cent right ear and complete absence of vestibular function in the left ear. Patient stated he was getting deafer and deafer. Up to the time of tonsillectomy the patient continued to have attacks of vertigo and increasing deafness. After removing the tonsils, the patient was immediately relieved in that he had no further attacks of vertigo and there has been no further progress in the loss of hearing for a period of one and a half years.

D. D.: The patient complained of a marked deafness following a dynamite explosion. His story was very convincing. He states that he had been unconscious for a while and had been actually knocked down by the explosion; that he had bled from both ears and that he was in bed for three weeks.

Repeated tests showed that he was malingering. He was naturally unable to reproduce the same chart each time. It became evident that although he probably did have a concussion of each cochlea, that the accident did not actually deafen him. First examination made it appear that he was entirely deaf in the right ear and had no appreciable hearing in the left ear, either. Vestibular function is normal. He blinked and looked in various directions when being examined, and in other ways gave evidence that he was in a nervous state.

It is perfectly possible that this man does have a cochlear defect, and that he may actually have the continuous tinnitus in both ears, of which he complains, but we are sure that he does not have a marked deafness—and we are also sure that he is lying.

SYMPOSIUM ON THE VIIIth NERVE.

III.—ARTIFICIAL AIDS TO HEARING.

VERN O. KNUDSEN, Ph.D., and DR. ISAAC H. JONES,
Los Angeles.

It is only natural that all of us should feel that in this mechanical and electrical age — this age of scientific achievement beyond anything the world has seen — we have bright prospects for mechanical and electrical wonders in artificial aids to hearing. As perfection has been more nearly attained in other electroacoustical devices, many of us naturally have the thought that all we need to do is to present our problem to the manufacturers of acoustical devices, and to expect from them the perfect instrument to help those with impaired hearing. The problem is definite. Compared with what has been accomplished in the field of general communication, such as long distance and multiplex telephony (in which at least five telephone conversations are transmitted over the same pair of wires at the same time), and transoceanic radio telephony, it would seem that ours should be a very simple problem. However, the problem is not so simple as it seems.

The purpose of this paper is to show that there is no complete solution to the problem of the deafened individual; that perfect instruments are not possible; that the defect in the individual may be of such a nature that an artificial aid cannot help him; but also to show not only what cannot be done, but, what is much more important, what can be done. Above all, we wish to show that the otolaryngologist has a new opportunity for service, in prescribing artificial aids to hearing.

In routine office practice, it is now possible to prescribe artificial aids for hearing, just as the ophthalmologist prescribes artificial aids for seeing. Recent developments in physiological acoustics now make possible this new service. In addition, there has been such an improvement in the performance of available hearing devices as to make it worth while to prescribe them.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 11, 1934.

In prescribing such aids, we now have dependable and quantitative methods of rating these instruments. For those who are chronically deafened, this surely is the most practical service that the otolaryngologist can render.

It is hard to understand why more artificial aids are not used at the present time. The instruments are now worthy of being used; the manufacturers have eliminated many of the unsightly features of the old types of instrument; they are less bulky and can be worked without serious inconvenience. It is most unfortunate that perhaps only one out of 50 persons who certainly should have an artificial aid actually has one. As otolaryngologists we can serve a purpose in encouraging our hard-of-hearing patients to derive the benefit which is now available to them in these artificial aids to hearing. Incidentally, with the more general use of instruments, which is highly probable, there will be less and less embarrassment to the individual in wearing the instrument. If only a few individuals wore eyeglasses, they might feel conspicuous; as it is, there is hardly any embarrassment to an individual wearing an artificial aid to seeing. The embarrassment that used to be felt in wearing aids to hearing was justifiable not only because they were worn by so few people, but because the instruments themselves gave such meager benefit. As one patient expressed it, "I never hesitated about putting on eyeglasses, because I accomplished an object; I saw things clearly. I did not purchase a hearing aid for years, because I could not hear clearly with it, and in fact I was more confused with it than without it. So why should I have purchased one?" Even today hearing aids are much more widely used than they were only five years ago, and they are not only worth using, but also they are much less conspicuous than they used to be.

In the popular mind there is an almost universal fallacy. People are afraid to use hearing aids because they have been told over and over again, "If you once use a hearing aid you will never be able to get along without it." This brings on the natural fear that their own unaided hearing will not be so good, if they once have become used to an aid. Although the idea is entirely false, it will probably take from 25 to 50 years to eradicate this concept. No doubt, this thought came from the experience that people have with eyeglasses.

It is true with the eye, but it is not true with the ear. When anyone has been forcing his eyes by straining his accommodation and convergence in order to see well, he naturally needs glasses. The glasses relieve this "eye strain." Because of this relief, the individual loses some of the power of accommodation; so when he takes off his glasses it is true that he is not able to see so well as he did before he began wearing the glasses. No doubt, this is why people think that the same thing applies to hearing. A hearing aid gives no such result. When the aid is used, the sounds are simply made louder. When the aid is taken away, the ear hears precisely as it did before the aid was used. Surely, there are thousands of individuals who do not use hearing aids because of this fear.

From exactly the opposite point of view, there is another false idea that is prevalent. Certain people teach that a hearing aid will "develop the ear." It is not the end-organ that is developed by the use of an aid, but the cerebral centres. The individual hears things that he did not hear before, and this naturally develops the central auditory areas.

In brief, if a precise diagnostic study shows that a patient would benefit from a hearing aid, the instrument never causes one to be "worse off," but always "does good."

It is probable that if the otolaryngologist does not assume responsibility and prescribe artificial aids for his patients, there will soon appear a new type of technician — the "audiometrist." Such a technician would naturally have a very limited knowledge of the diagnosis, and evidently would be unaware of ear, nose, and throat, and general conditions, most important to the best interests of the patient. In other words, just as the ophthalmologist has had the problem of the optometrist, so it seems that a similar problem will very soon enter the field of otolaryngology.

The oldest forms of artificial aids for hearing are the ear trumpet and the speaking tube. These two types of instrument essentially place the listener nearer the speaker; in the case of the horn by presenting a larger opening to the source of the sound waves, and in the case of the speaking tube by conveying a large portion of the speech energy directly into the listener's ear.

The horn or trumpet is the most primitive type. There are two physical principles involved in the use of the horn. It acts as an amplifier because the outer end of the horn can collect a greater amount of sound energy than the ear, unaided. Again, the horn acts as a resonator, in a manner somewhat similar to an organ pipe. It used to be thought that if the opening at the big end of the horn were, for example, a hundred times larger than the opening at the small end, one might expect an amplification of one hundredfold. Such an amplification, however, is not realized. Much of the sound energy is lost by reflections from the sides of the horn or from the open end of the horn; also the different components of the sound interfere with each other. The amplification is, therefore, much less than one hundredfold. Also, because the horn acts as a resonator, certain frequencies are emphasized more than others, and as a result distortion is introduced. This distortion produces a "tinny" or metallic quality to the sound. However, the horn, or trumpet, is of considerable benefit to certain patients with perceptive impairment, particularly as the horn is free from the adventitious noises produced by carbon granule microphones.

Shortly after the advent of the telephone, aids were designed which utilized the component parts of a telephone system; a carbon granule microphone connected to a battery and a telephone receiver. In the more recent models of this type of instrument a microphonic amplifier has been introduced between the microphone and the receiver. To this day, this type of instrument is the most practical, simply because of cost and portability.

The advent of the thermionic vacuum tube, so commonly used in our radios, has made possible an almost unlimited amplification of sound. The vacuum tube, in fact, marked the beginning of a new epoch in the amplification of sound; it made possible an amplifying device which was a phenomenal improvement over the carbon microphone. Instruments with vacuum tubes are now available, but most of them are too heavy and cumbersome to be conveniently carried.

A recent development which satisfactorily meets the requirements for the use of the telephone by most hard-of-hearing persons has been designed by telephone engineers. The device can be attached to any telephone. This instrument

consists of a separate vacuum tube amplifier inserted between the incoming line and the telephone receiver. The unit provides an amplification of 20 to 30 decibels over a wide range of frequencies, and has an adjustable volume control so that the hard-of-hearing person may adapt it to his own loudness requirement. The instrument is not sold but is leased on a monthly rental basis by the telephone company.

The ideal aid would consist of the following: *1.* A microphone, free from distortion and adventitious noise; *2.* a vacuum tube amplifier, providing the required amount of amplification, without distortion and free from adventitious noise; *3.* a receiver which reproduces all audible frequencies without distortion; *4.* sufficient separation between the microphone and the receiver to prevent "howling." When the receiver is too near the transmitter, the sound emitted by the receiver is naturally again communicated into the transmitter; this process is repeated and gradually builds up to such an extent that the circuit "howls." To be ideal, the instrument must be used in a room which is free from excessive reverberation, noise, and other acoustical defects.

All of this is obtainable today. Unhappily, we cannot prescribe such an instrument because it costs too much and it could not be made sufficiently portable. Although an entirely perfect microphone, or transmitter, has not yet been made, it is possible to secure a very good one — of the electrodynamic or ribbon type — for about $150. A first class amplifier costs no less than $150. These nearly perfect transmitters and amplifiers are actually obtainable. Exçellent receivers are also available, but such receivers are not on the open market, although laboratory instruments have been made which are entirely satisfactory, and which could be marketed for about $25. Allowing for the assembling of these three parts, the price of such an almost perfect instrument would be over $400. As to portability, it would be very difficult at the present time to compress this equipment into a bulk of much less than one-half cubic foot, and to reduce its weight below 25 pounds.

Even if the patient could afford such an instrument and could be able to transport it, it would still be difficult to provide the required separation of the microphone and receiver— as a separation of at least 20 feet is necessary in many cases to prevent howling. Naturally, the practicing otologist has

little conception of the intricacies and difficulties which restrict the production of such an ideal instrument. Nevertheless, it is not unreasonable to anticipate great improvements in the present equipment, and we may look forward to better service from artificial aids to hearing — contingent, of course, upon the meeting of the requirements that we have just outlined.

Curiously enough, the otolaryngologist is better acquainted with the use of artificial aids for seeing than for hearing. In our attempt to understand the problem of artificial aids for hearing it is helpful to compare the ear with the eye. Both hearing and seeing are made possible through the detection of vibratory impulses by the nerve filaments of the cochlea or the retina. To this extent there is a similarity between the ear and the eye. The great difference lies in their conductive mechanisms. The external and middle ear constitute an acoustical transformer. Such a transformer is necessary: the external vibrations are in a gaseous medium, the air; these impulses are received in a liquid medium, the endolymph of the cochlea. If sound waves originate in air and strike a liquid, more than 99.9 per cent of the sound energy is reflected from the surface of the liquid, and less than 0.1 per cent penetrates the liquid itself. Although we do not know precisely how efficient the middle ear mechanism is in its transformer action, we do know that it is so designed that it meets the physical requirements for transferring energy from air to liquid. In the language of physics, it "matches the impedences" of the air and the cochlear liquid; in other words, it meets the requirements of efficient transmission of sound energy. The chief function of the middle ear is to convert a pulsation of relatively large amplitude and small force into one of relatively small amplitude and large force.

The eye is different. Instead of an energy transformer, the conducting mechanism of the eye — which is transparent to the incoming light waves — simply focuses these light waves upon the retina. The focusing effect depends only upon the indices of refraction and the curvatures of the component parts. It is not an energy transformer, like the middle ear, but performs the simple function of refraction.

Consequently, in pathologic lesions, whereas there is a similarity between the cochlea and the retina, there is a dissimi-

larity between the lesions of the conducting mechanisms of the ear and of the eye. A nerve lesion of either organ limits the reception of stimuli. No artificial aid can completely compensate for the deficiency caused by such a lesion, whether in the ear or in the eye. On the other hand, artificial aids can do much to make up for the deficiencies due to lesions in the conducting mechanisms, both in the ear and in the eye. In the ear, the deficiency can be overcome by suitable amplification of sound; in the eye, by suitable refraction of light. Eyeglasses simply refract light waves; that is all that is expected of them. If an eye has ulcers of the cornea; opacity of the fluid in the anterior chamber; a cataract of the lens; or opacity of the vitreous — all the eyeglasses in the world could not overcome such "conductive" defects. In fact, no one would ever expect such a service from eyeglasses. In practice, no one asks for eyeglasses which will overcome opacities; it is universally known that vision is impossible through an opaque substance. Consider similar lesions in the ear: atresia of the external canal; thickening or stiffening of the drum membrane; arthritis or fibrosis of the ossicular chain; or fixation of the stapes in the oval window—such lesions are analogous to the lesions of the conductive mechanism of the eye. Fortunately, although so much is expected in overcoming the effects of such lesions in the ear, yet this very thing is possible. We cannot transmit light through the conductive lesions of the eye by the amplification of light, but we actually can transmit sound through the conductive lesions of the ear by amplification of sound. In this respect, artificial aids for the ear can accomplish more than artificial aids for the eye. The general concept has been that glasses are very satisfactory, and that aids to hearing are very unsatisfactory. The reason is obvious. The glasses do accomplish very well their simple purpose of refraction. But they do so only if the eye is functioning both in its conductive and perceptive parts. In the case of an ear defect, we are fortunate in not having the simple problem of refraction as we have in the case of an eye defect; but, on the other hand, we are fortunate in being able to overcome the effects of conductive lesions in the ear, by suitable amplification, which cannot be done for the eye.

The prescribing and use of artificial aids to seeing have become an established and important part of ophthalmologic practice. The science of optics developed earlier and engaged

the attention of many more scientific workers than did the science of acoustics. Not only has the nature of the errors of vision been well understood for centuries, but artificial aids for impaired vision have long since reached a high degree of perfection. It is true that the nature of hearing defects also has been known for centuries, but even the approach to the scientific design of artificial aids to hearing is of comparatively recent date, almost as if it were a matter of yesterday.

Most of the defects of the refractive mechanism of the eye are well understood, and are now capable of precise measurement and correction. The correction of these visual defects usually involves a change of focal length; it does not, as a rule, involve the matter of amplification of the intensity of light. Further, since the eye responds to wave lengths that are almost infinitesimal in length, it is possible to use relatively small lenses for the correction of refractive errors. Astigmatic and aberrational correction can be accomplished by exceedingly minute alterations in the curvature of the lens.

The ear, on the other hand, regarded as a physical instrument, presents many unsolved difficulties. All devices which have been invented for amplifying sound are limited in the extent to which they amplify, and the portable devices which have been adopted for hearing aids introduce serious distortion.

There is another disadvantage in the prescribing of aids to hearing. It is true that we are able to measure deficiencies of hearing with as high a degree of precision as the ophthalmologist attains in measuring deficiencies of vision. The examination and tests can reveal the diagnosis for the ear with the same precision as for the eye. However, the ophthalmologist is able to write his prescription on a piece of paper, with the knowledge that the "prescription will be filled" in every detail. The otolaryngologist cannot do this. There are as yet no available manufacturers or technicians to fill the prescriptions for aids to hearing. Our situation is similar to what it would be for the ophthalmologist, if, after testing his patient, he had to choose from a limited number of stock eyeglasses.

Having in mind these analogies and differences of hearing and seeing, we can better appreciate the requirements for a suitable aid to hearing. If the cochlea be normal, appropriate

amplification alone can restore hearing to the normal. If, however, the receptive mechanism of the cochlea be degenerated, the benefits of amplification are naturally limited. Any such amplification produces an overstimulation of the cochlea, with a resultant irritation and confusion, which naturally defeats its own purpose. Given a slight cochlear defect, only a moderate amplification is required. This will be readily tolerated, and the hearing will be improved. Given a gross cochlear defect, a high degree of amplification is required. This will not be tolerated, nor will it contribute to the intelligibility or quality of speech and music. Therefore, it is in cases of conductive defect, that amplification produces the best results, because a high degree of amplification is possible without overloading the cochlea. In cases of cochlear lesion the benefits to be obtained by an artificial aid are restricted by the degree of excitation which the cochlea can tolerate.

When the average patient comes to us and states that he has difficulty in hearing speech or music, we realize, even before examination, that he probably has less than 65 per cent of hearing. In order to restore this patient to normal hearing, by means of an aid, it is necessary to provide a high degree of amplification. For example, if the patient has 65 per cent of hearing — that is, a hearing loss of about 40db. — it would be necessary, in order to restore his hearing to normal, to provide an energy amplification of ten thousandfold.

Let us consider the case of Sidney M. R., whose chart is shown in the preceding paper. This chart shows that the patient's hearing is nearly normal by bone-conduction, which indicates that the defect is almost entirely conductive, and therefore can be entirely corrected by suitable amplification. This patient exhibits a hearing loss of approximately 50 db. throughout the important range of frequencies used in speech and music. Consequently, an artificial aid which would completely correct this defect would need to amplify all frequency components of speech and music approximately 50 db., or one hundred thousandfold. This is the amount of amplification which would be required if we lived in perfectly quiet surroundings. As a matter of fact, this patient would use his artificial aid under more or less noisy conditions, so that such a high degree of amplification would not be at all necessary. In the average room, in which there is always

some noise, the person with normal hearing is bothered by the noise, and consequently speaks louder than he would in a quiet place, thus rendering it unnecessary to have such a high degree of amplification in the hearing aid. The amount of noise in the average room amounts to about 30 db., which would reduce the requirements of amplification from 50 db. to 20 db., or an energy amplification of only one hundredfold, an amount which can readily be attained by a well designed aid.

Consider also the left ear of Joseph Br. In this case, at 128 cycles, the hearing is 40 per cent, or the hearing loss is 55 db. At 512, the hearing is approximately 70 per cent, which corresponds to a hearing loss of 38 db. And at 2048, the hearing is 40 per cent, which corresponds to a loss of 77 db. Consequently, in order to restore this patient's hearing curve to the 100 per cent norm, it would be necessary to provide an amplification of 55 db. at 128, 38 db. at 512, and 77 db. at 2048; that is, the amplification should be selective and not uniform for all frequencies. One would naturally suppose that such a selective type of amplification would meet the requirements for the most satisfactory aid. Ten years ago we so regarded the matter, and designed and constructed some artificial aids for hearing along this principle of selective amplification. However, our early experience revealed that we were dealing with a problem in cerebration as well as one in the physical amplification of sound. The individual with an impairment of hearing is accustomed to hearing sounds as they are communicated to him through his defective end-organ. Perhaps this explains the statements that the patients made: they heard as well by uniform amplification as they did by selective amplification, even though the selective amplification was of the type required to elevate all frequency components appropriately; that is, to meet the deficiency at each frequency, as shown on the audiometric chart. However, we still feel that selective amplification is not merely a theoretical ideal: it is probable that with continued use of such amplification, individuals with impaired hearing would come to adapt themselves to the appropriate type of amplification, and thus properly interpret normal auditory impulses. This would give the patient the same auditory impulses that the normal receives. Certainly, the ideal would be not only to send in normal impulses at all frequencies, but to have the

patient re-educated by these normal impulses. At the present time, however, selective amplification has not been generally adopted by the manufacturers of artificial aids. Further investigation of this problem should prove of real value. One of the writers has suggested this problem to one of his students who is now conducting a series of experiments on the use of selective amplification for hard-of-hearing persons.

H. M. Wharry[1] reports that he has fitted a number of patients, according to prescription, with hearing aids which provide selective amplification. By utilizing both hard and soft carbon diaphragms, he is able to vary the resonant frequencies and thus emphasize the amplification of selected frequencies. Such instruments have proved more satisfactory than those which are not made according to prescription. However, he makes the qualifying statement that "further improvements can no doubt be made."

The question is often asked, what patients can benefit from a hearing aid? In the first place, many individuals who have 80 or only 75 per cent of hearing do not notice in any way that they are becoming hard-of-hearing. As we have already emphasized, this is due to the noisy conditions under which we live; ordinary conversation actually is spoken on a sufficiently loud level to be understood about as well by a person with 75 or 80 per cent of normal hearing as it would be by a person with normal hearing, simply because a person with normal hearing is brought down to a 75 or 80 per cent level by reason of the masking effect of the noise. When a person's hearing is between 75 and 60 per cent, on the average, he can hear if those who speak to him speak clearly and with only slightly more than average loudness. If a patient has a hearing of 60 per cent or less he should certainly consider the use of an artificial aid. In general, a patient should use a hearing aid just as soon as he or she finds difficulty in hearing well in the ordinary conditions of life. In fact, all those whose hearing is between 20 and 75 per cent should become potential users of an aid to hearing. In a purely conductive lesion we are dealing with a normal cochlea, and all that is needed to restore hearing to normal in such an individual is appropriate amplification.

A conductive lesion cannot reduce the hearing below 50 per cent. A normal cochlea imbedded in the skull, without

any conductive mechanism whatever, would show a hearing acuity of about 50 per cent; the only loss being that due to the insulation of sound of the surrounding soft tissues and skull. Even six inches of concrete reduces the intensity of sound transmitted through it only 55 db. Therefore, if any individual has a hearing of less than 50 per cent, that is, a hearing loss of more than 55 db., we know at once that at least that portion of the hearing loss which exceeds 50 per cent must be of the perceptive type.

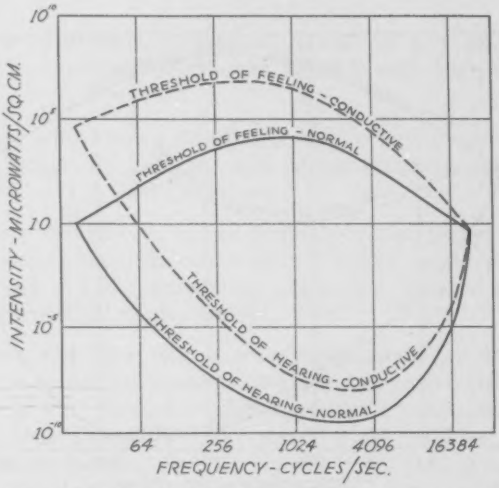


Fig. 9. Showing the "auditory area" for the normal and for a patient with purely conductive impairment.

The nature of conductive and perceptive lesions, in respect to the use of hearing aids, can be better understood by referring to the two charts given in Figs. 9 and 10. The solid line curves in Fig. 9 give the lower and upper threshold limits of hearing in the case of the normal. The dotted lines give the lower and upper threshold curves for a typical case of conductive lesion. It will be noted that both the lower and upper threshold curves for the conductive are shifted upward the same amount. (In gross conductive lesions the upper threshold curve may not be elevated quite so high as is indicated in Fig. 9, but the curves emphasize the important fact that the full range of a normal cochlea can be utilized.) In other

words, the total auditory area is the same for the conductive as it is for the normal, and therefore all that is needed to restore this individual to normal hearing is adequate amplification.

Fig. 10 shows the hearing chart for a typical case of perceptive impairment. This chart differs in two important respects from the chart for the conductive. In the first place, only the lower threshold curve is shifted upward; the upper threshold curve remains the same as the upper curve for the

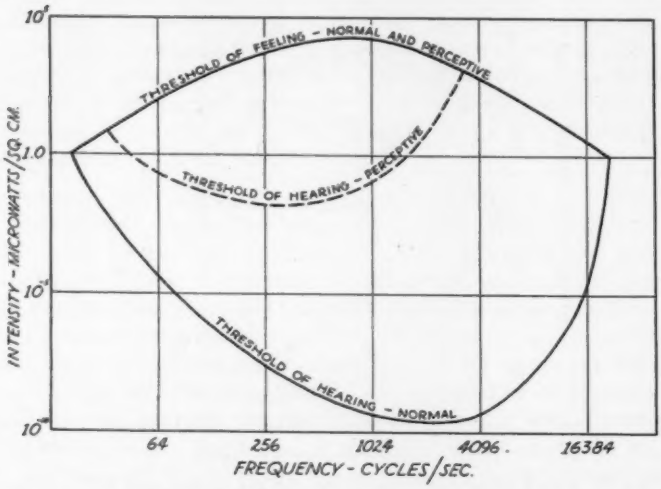


Fig. 10. Showing the "auditory area" for the normal and for a patient with purely perceptive impairment.

normal. In the second place, the hearing loss at high tones for the perceptive is very much greater than it is for low tones. The person with perceptive impairment, therefore, not only has a much smaller auditory area for the perception of the sounds of speech and music than does the person with normal hearing, but the area is most limited for the higher frequencies which are so important for the understanding of speech, especially the consonants. It is obvious, therefore, that a person with a 50 per cent perceptive impairment will not derive so much help from an artificial aid as will a person with a 50 per cent conductive impairment. Again, in the case

of the person with a 50 per cent conductive impairment, there is this extensive area between the lower and upper threshold curves. It is therefore possible to provide a large amplification without reaching the upper level of the disturbing and painful. In the case of the perceptive, on the other hand, such a large amplification is not possible, because this amplification soon reaches an intensity which is both disturbing and painful.

In brief, in answer to the question as to which individuals can receive benefit from an artificial aid, there are four classifications:

1. Those who have conductive lesions of any magnitude whatever can receive the greatest possible help from an artificial aid.

2. Those who have a purely perceptive impairment which does not exceed 50 per cent will derive sufficient help from an artificial aid to justify its use.

3. Those who have a mixed conductive and perceptive defect, in which the conductive defect might be as great as 50 per cent and the perceptive defect not greater than 30 per cent, will also receive benefit.

4. Those who have only a perceptive defect, but in excess of 50 per cent, cannot expect to derive much satisfaction from an artificial aid. In fact, a high degree of amplification, dangerously near the painful threshold, could produce further damage to the cochlea. In other words, if the patient has more than 50 per cent of cochlear function he is reasonably safe in using an artificial aid, provided the amplification be not too great. The aid should be used with caution so that the already weakened cochlea is not overstimulated.

When a hearing loss is as great as 60 per cent, owing to perceptive defect only, the residual hearing for frequencies above about 1,000 cycles is so meager that average speech, even when amplified, is so badly distorted as to become almost wholly unintelligible, and the disturbance, owing to the increased intensity of the pulsations reaching the cochlea, in the case of high amplification, offsets any value from the amplification.

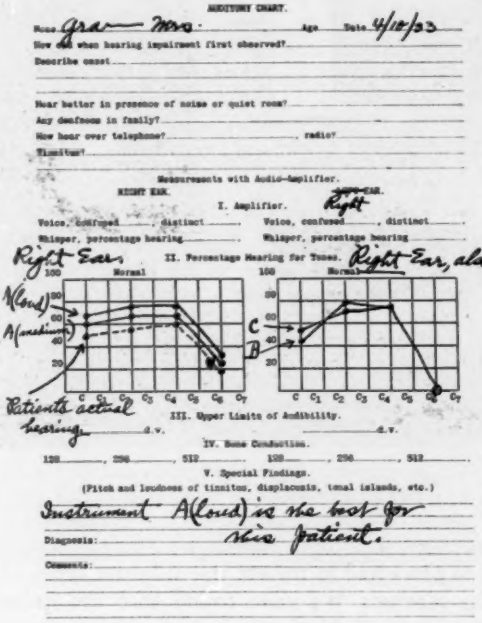
If the audiometer with which the patient's hearing is tested be equipped with a microphone, as we have in our audio-

amplifier, it is so easy to determine and demonstrate to the patient at the time of examination, whether he will or will not hear well with an artificial aid. Further, if the audiometer be equipped with electrical filters so that one can, at will, produce any degree of amplification for different frequencies, it is possible also to find out if the patient can derive benefit from any type of selective amplification. In brief, the question as to whether he should have an artificial aid is settled for him, at the time of the examination, without reference to the recommendations of those who have instruments for sale.

The degree of help which a patient will derive from an artificial aid depends, first, upon the nature of his hearing defect and, second, upon the characteristics of the hearing aid. As Dr. Fletcher[2] has shown, it is possible to determine quantitatively a figure of merit for any patient. It is necessary to know: *1.* the audiogram for the patient, and *2.* the amplification curve for the instrument. Dr. Fletcher has prepared a table which gives the contributions of the various frequency bands to the figure of merit of the instrument. This method of determining just what benefit any patient would receive from any particular aid naturally depends upon our having available the amplification characteristics of the various instruments. Unfortunately, at the present time, it is not possible to obtain the amplification curves for the instruments on the market.

It is highly desirable that manufacturers should furnish with each type of hearing aid they make a certificate setting forth: *1.* the amplification characteristics of the instrument, and *2.* the noise audiogram of the instrument. These data should be furnished at least for frequencies of 128, 256, 512, 1024, 2048 and 4096 cycles. Again, there is not only a difference in the amplification characteristics of the different makes of instruments, but there is a variation in the amplification characteristics of instruments bearing the same trade name. Therefore, it seems to us that the immediate practical solution is for the otolaryngologist to make a careful diagnosis, including precise hearing measurements, and then to repeat these hearing measurements with several of the best makes of instrument on the market. In this way each patient receives specific and intelligent guidance in the purchase of the best instrument to suit his individual need.

Although amplification curves are as yet unavailable for most artificial aids on the market, such curves have been obtained for some of the instruments. In Fig. 2 are shown such amplification curves. In general, those aids which show the least amplification are those produced as long as ten years ago; whereas, those which show the greatest amplification are recent products. These charts, therefore, show not only the


AUDITORY CHART.

Name Gra— Mrs. Age Date 4/12/23

How old when hearing impairment first observed?

Describe onset

Hear better in presence of noise or quiet room?

Any deafness in family?

How hear over telephone? radio?

Tinnitus?

Measurements with Audio-Amplifier.

RIGHT EAR. LEFT EAR.

I. Amplifier. Right

Voice, confused , distinct . Voice, confused , distinct .

Whisper, percentage hearing . Whisper, percentage hearing .

Right Ear, II. Percentage Hearing for Tones. Right Ear, also.

Normal 100 Normal 100

A(loud) A(medium)

Patient's actual hearing

C B

C C1 C2 C3 C4 C5 C6 C7

III. Upper Limits of Audibility.

d.v. d.v.

IV. Bone Conduction.

128 , 256 , 512 128 , 256 , 512 .

V. Special Findings.

(Pitch and loudness of tinnitus, displacusis, tonal islands, etc.)

Instrument A(loud) is the best for this patient.

Diagnosis:

Comments:

characteristics of various instruments, but also reveal the progress which has been made in the development of such devices.

It is not our purpose to list all the aids to hearing that are now available on the market. Of course, there are scores and scores of "aids" that are downright fakes. The advertisers of such fakes simply prey upon the credulity of the afflicted. In sharp contrast, there are numerous manufacturers who are making an earnest and scientific attempt to provide

useful aids. There are available today many instruments, more or less comparable with each other, which incorporate the best principles and devices that are known today. In the portable type that is worn on the clothing the amplifier unit is of the microphonic type, which, of itself increases the amplification about 10 db. between frequencies of about 500 and 3000 cycles.

Fig. 11. The speech articulation test, for vowels and consonants, without the use of a hearing aid.

At the present time it is customary for the patient to go from place to place and to try out the different types of instrument on the market. We have found that the judgment of the patient is not reliable in the selection of an instrument, particularly as this judgment is naturally influenced by sales arguments. It is only by actual tests, with precision measurements, that the real facts can be obtained.

This queston is so often asked, "What can be done, in order to prescribe the best artificial aid for any given patient?" In answering this, we shall discuss a typical case (Mrs. Gra--). The chart shows, in dotted lines, the curve of the patient's hearing in the right ear, without the use of an aid. The four solid line curves represent the hearing acuity with the use of four different instruments. The patient wears

the artificial aid, and the transmitter is held against the receiver of an audiometer. If the instrument is of any value this second test will indicate a smaller loss of hearing than was indicated by the original chart. Although the differences between the original and the charts obtained with the instruments do not give the amplification characteristics of the various aids, they give us a quantitative measure of the value that each instrument would have for this particular patient. Thus, it may be found that some instruments will provide a relatively large amplification for a wide range of frequencies, whereas other instruments may show an amplification over

Fig. 12. The speech articulation test, for vowels and consonants, with the use of a hearing aid.

a more limited or even a different range of frequencies. It will be noted that instrument A (loud) provides a slightly better amplification characteristic than either A (medium), B, or C, particularly at high and low frequencies. This demonstrated that Mrs. Gra-- heard best with instrument A (loud). This was corroborated by the speech articulation tests which are described in the next paragraph.

The speech articulation tests are similar to those employed by telephone engineers in testing the efficiency of speech transmission. The speaker, or caller, calls out a series of

monosyllabic words. The patient, four feet away from the caller and in a position where the lips of the caller cannot be seen, listens to these words and either writes down what he hears, or calls back each word as he hears it. A record is kept of the number heard correctly or incorrectly. The caller must practice this method in order to minimize the errors which occur from varying the loudness or quality of the speech sounds. It may prove advisable to have phonograph records made, which would overcome such errors. This would make the speech articulation that much more precise. One list of words is used for the vowels and another list is used for the consonants. These lists are similar to those prepared by the Bell Telephone Laboratories.

VOWEL LIST.

bat	cake	buck	rake	right
hit	naught	bought	sack	weep
boat	rut	seem	rut	bed
meat	soup	pick	back	hate
balk	doubt	bait	bit	heat
look	cup	bake	let	gout
peck	tight	tight	greet	type
goat	hook	rout	bet	book
rip	puck	pig	reap	sit
bite	rub	red	sought	rat
seat	but	soap	mike	set
boot	talk	fought	bout	hut
state	pit	dime	hub	beak
met	loop	caught	mat	cat
kite	pat	site	coal	leak
nut	cut	moon	took	ripe
cough	take	coat	pouch	neat
rope	bike	mate	soon	pack
pout	pet	taught	hope	bide
rat	good	rot	rate	bean

A sample list of articulation words is shown in the accompanying table, for the testing of vowels. A similar list is shown for the testing of consonants. On the chart, the consonants to be tested are printed in italics. In the vowel list only errors in the vowels are recorded. Thus, if "bite" is heard as "bike" it is counted as correct, since the vowel is heard correctly. If these 100 words are used in the tests for the vowels the number heard correctly gives the "percentage vowel articulation."

CONSONANT LIST.

by	*she*	*foe*	*why*	*show*
die	*lick*	*tick*	*tie*	*ran*
guy	*bill*	*nay*	*thy*	*beach*
high	*night*	*bee*	*sigh*	*fee*
lie	*pit*	*fay*	*bust*	*cog*
whim	*hit*	*wag*	*gay*	*pick*
win	*hash*	*ten*	*lay*	*hill*
pie	*cliff*	*when*	*tin*	*no*
with	*jazz*	*biff*	*pan*	*ring*
whip	*toe*	*wrath*	*see*	*reach*
wiz	*thee*	*which*	*hoe*	*log*
vie	*saw*	*fie*	*lee*	*fill*
thigh	*bun*	*wig*	*rim*	*rich*
shy	*dare*	*wick*	*fin*	*day*
wry	*gate*	*will*	*dish*	*go*
day	*ham*	*nigh*	*bath*	*thin*
hay	*low*	*wing*	*tip*	*vine*
him	*ram*	*wit*	*which*	*fizz*
sing	*sin*	*wish*	*vane*	*rip*
row	*put*	*whiff*	*thick*	*rich*

Returning to the case of Mrs. Gra - -: Without the use of an artificial aid she heard 50 out of the 100 vowel words. She therefore has a vowel articulation of 50 per cent. Similarly, 100 consonant test words were used, and the number heard correctly constituted the percentage consonant articulation. Without an aid, she showed a 26 per cent consonant articulation. With instrument A, her vowel articulation was 94 per cent and her consonant articulation 41 per cent. With instrument B, the vowel articulation was 82 per cent and the consonant articulation 32 per cent. With instrument C, the vowel articulation was 92 per cent and the consonant articulation 38 per cent. As will be seen, the articulation for both vowels and consonants is best with instrument A. This is consistent with the audiometric findings. Consequently, instrument A was recommended for Mrs. Gra - -. Although this method occupies a great deal of time, it provides a means of determining which instrument any given patient should purchase.

Unfortunately, there is one other deduction that may be made from the results of such tests; you will observe that even these instruments used on Mrs. Gra - -, which are three of the best instruments that are now available, provide only

limited help. Thus, Mrs. Gra - -, with the use of instrument A, shows an improvement from 50 per cent to 94 per cent for the vowels, and from 26 per cent to 41 per cent for the consonants. This instrument, therefore, falls far short of what is desired in respect to the consonants — so essential for the recognition of speech.

Similarly, we note that the tests with the audio-amplifier show an improvement of only 20 per cent at 64 cycles; 21 per cent at 256 cycles; 18 per cent at 1024 cycles; and 18 per cent at 4096 cycles. In other words, the amplification did not, by any means, bring the hearing to normal. This, of course, is somewhat disappointing, and can be improved only by using a larger and more expensive instrument, containing vacuum tubes. Such an instrument, however, is not only expensive, but would be too heavy to be readily carried about by the patient. However, the benefit derived from the small and readily portable aid is appreciable. The hearing of nearly all the vowels and a few extra consonants means to this patient all the difference between the hearing and not hearing of conversation of average loudness. Therefore, from the practical standpoint, such an instrument is far more valuable to the patient than the mere numerical findings would seem to indicate.

SUMMARY.

1. There is no complete solution to the problem of the deafened individual, whether by suitable early treatment or by the use of an artificial aid.

2. We now have dependable and quantitative methods for rating the performance of artificial aids. If we do not accept this responsibility, others will.

3. By means of these methods, it is possible for the otolaryngologist to prescribe artificial aids for hearing, as the ophthalmologist prescribes artificial aids for seeing.

4. It is almost certain that there will be more and more improvements in artificial aids, and it is not improbable that there will be radical changes in basic principles in the approach to this problem, as well as in the design of the instruments. We can trust to the awakened interest in the subject of physiologic acoustics, and the continued improvements which

are being made in communication instruments to insure a brighter future. However, we should surely not wait for further developments. Only too few of our patients come to us early enough to enable us to prevent them from becoming deafened. A large majority of the patients who come to us with a hearing defect would be benefited by the very artificial aids that are now available. To prescribe the proper artificial aids for those who are chronically deafened is the most practical service that the otolaryngologist can render.

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1930 Wilshire Blvd.

SYMPOSIUM ON THE VIIIth NERVE.

IV.—NEURO-OTOLOGIC STUDIES IN EPILEPSY.

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It may seem far afield, on first thought, to study epilepsy from the standpoint of the ear. However, as nystagmus is so frequently seen during an epileptic attack, it may have occurred to many clinicians, familiar with vestibular study, that there might be some relationship between epilepsy and the vestibular apparatus. It was with this thought in mind that a series of 35 epileptic patients was studied, purely from the standpoint of the ear and VIIIth nerve, hoping that some light might be shed on the possible involvement of the brain stem and basal ganglia.

The first work along this line was set forth in a report by I. H. Jones, in 1923. Twenty-seven cases had been examined, all private cases that had been carefully studied by neurologists and neurologic surgeons. On tabulation of the findings no characteristic condition was discovered in the nose, throat, external ear, or cochlea; nor was there any suggestion of intracranial auditory involvement. In contrast, vestibular impairment or abnormality was found in nearly all the cases. Of the 27, only three showed the normal constitutional responses to the vestibular tests. They failed to show the usual pallor, sweat and nausea that one finds in the normal after prolonged turning and douching.

In 1928, J. S. Robinowitsch, of Odessa, who had followed up this work, reported 86 cases. His cases included all types of epilepsy, not limited to the so-called "idiopathic" type. His cases included many of the Jacksonian type. His findings showed a greater percentage of normal findings than in the series reported by Jones, and he did not observe such a striking lack of constitutional responses. In the main, however, his findings were similar, and led him to similar conclusions. As quoted from Jones, "The phenomena suggests that the fairly

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 15, 1934.

well known vestibular mechanism is connected with the unknown mechanism through which epileptic phenomena are produced."

An interesting observation on the central tracts of the vestibular nerve was reported by Jones and Spiller, in 1925. Their case was one of epidemic encephalitis in which there was complete absence of vestibular function with no impairment of auditory function. The case presented a spontaneous nystagmus of the type suggestive of a central vestibular lesion. Autopsy revealed nothing visible to the naked eye, but on making serial sections and staining, there was found a limited area of complete degeneration in the center of the tegmentum, extending from the lower part of the medulla oblongata well into the lower part of the pons with the raphe as its center.

It was their conclusion from this study that all or nearly all of the central tracts concerned with vestibular function pass through the area represented by this degeneration and that few of the fibres of the cochlear system are present in this region.

Epilepsy has long been looked upon as a disease of the cerebral cortex, but recently neurologists have come to consider that at least a phase of the seizure probably arises in the brain stem, or basal ganglia.

The following report is based upon the studies of 35 cases of proven epilepsy of the so-called idiopathic variety, the cases being taken from a neurologic service of the Los Angeles General Hospital and referred to the Ear Clinic, Department of Neuro-Otology, under the supervision of Dr. I. H. Jones. All had general physical examinations, followed by the neurologist's special report, and all with a history of head injury or suspected intracranial neoplasm were excluded. None were included that gave a positive Wassermann.

Before proceeding with the actual analysis of the cases, it might be well to review the methods of examination used, and a brief outline of the nerve pathways and their function.

The examination includes that which we are pleased to call the American technique, which, briefly stated, consists of: *1.* Examination for spontaneous phenomena; *2.* turning tests; *3.* caloric or cold douching test.

Examination for Spontaneous Phenomena: The patient was seated in a good light and the eyes carefully examined for

nystagmus by having the patient follow the examiner's finger, held about 12 inches away, moving it first in the vertical plane and then in the horizontal.

A few jerking movements of the eyes when looking to the extreme right or left in the horizontal plane may be considered physiological, but any sustained nystagmus in the vertical plane must be considered pathological. Spontaneous past-pointing was next looked for by having the patient with eyes closed, raise his hand above his head and then come down and touch the finger. Any past-pointing was carefully recorded. The patient was now asked to stand and any tendency to fall or sway was recorded.

Turning Tests: These were made with the patient seated in the usual type chair, with the head inclined 30° forward and the eyes closed. The patient was turned first to the right, making ten complete turns in 20 seconds, and observing the character and length of time the nystagmus continued after the chair was stopped. The patient was then turned to the left and the same procedure carried out. These findings were then recorded as after-turning nystagmus. The patient was then turned more rapidly, the rate being ten turns in ten seconds, and on stopping the chair the patient was asked to indicate in which direction he felt he was moving, by saying the words "right" or "left," and continuing to say them as long as he felt he was turning. This time was measured by the stop watch and recorded as after-turning nystagmus.

Statistical studies of 40,000 aviators selected at random and recorded in the *Air Service Medical* for 1918, placed the after-turning nystagmus when turning to the right as 23.5 seconds and when turning to the left as 23.2 seconds. Before these statistics were obtained the time had been considered as 26 seconds, but since then we have come to consider 24 seconds as the normal average.

There are no statistics available for the after-turning motion-sensing and inasmuch as this is a subjective rather than an objective response, a greater variation in findings might naturally be expected, but we have come to feel that the average duration of the vertigo is about the same as the time for the after-turning nystagmus, namely, 24 seconds.

The *caloric or douching tests* were made with the patient sitting in the turning chair with the head inclined 30° for-

ward, and eyes open with the gaze fixed on a spot on the floor ten feet away, water at a constant temperature of 68° F. was allowed to flow under gravity into the ear canal, striking the drum with gentle force. This was allowed to continue until a good response was obtained, or in case of no response the douching was continued for four minutes. The average normal response consists of a rotary nystagmus coming on in 40 seconds and continuing for two to four minutes. The head was now placed 90° back, bringing the horizontal canals in the vertical position. The nystagmus now changes from a rotary to a horizontal, the direction being toward the opposite side from the ear douched. While in this position the past-pointing was taken. The normal patient should past-point from six to 18 inches to the right, if the right ear was douched, or to the left if the left ear was douched. The patient was now made to stand and the direction of falling was noted. The normal patient should fall in the direction of the side douched.

After both the turning and douching tests such constitutional responses as pallor, sweat, nausea or vomiting were carefully noted.

It may be stated here that all the above tests were carried out at a single appointment, a procedure which cannot be recommended in any case except those with subnormal reactions.

It might be well at this time to briefly review the anatomical pathways and their distribution, and also the mechanism by which stimuli arising in the semicircular canals produce the phenomena of nystagmus, vertigo, past-pointing and falling. The concept that we have up to this time concerning the exact anatomical pathways and their distribution is based upon autopsy findings of lesions located in the region of the brain stem and basal ganglia. It may be hundreds of years before the exact histological distribution can be definitely proven.

The vestibular labyrinth is composed of three semicircular canals, two vertical and one horizontal, so placed as to occupy three separate planes in space. The VIIIth nerve is composed of a cochlear portion and a vestibular portion, and it is with the vestibular portion that we are most concerned in this study. Nerve fibres from each of the three semi-circular canals

go to make up this pathway which enters the brain stem at the junction of the pons and medulla. After entering the brain stem the fibres separate and take two distinct paths, those from the two vertical canals continuing together and functioning as one canal, while the fibres from the horizontal canal continue on a separate course.

In order to make this distribution more clear, each pathway may be represented as a capital "Y"; one arm of the "Y" eventually goes to the eye muscles and the other to the cerebellum. The tract to the eye muscles constitutes the vestibulo-ocular pathway; impulses transmitted along this tract produce nystagmus.

The tract to the cerebellum is known as the vestibulo-cerebellar tract, and impulses transmitted along this tract produces motion-sensing.

With this general distribution in mind, we may now trace the tracts more in detail.

The horizontal canal fibres begin in the crista of the horizontal canal, enter the cerebral cavity as a part of the VIIIth nerve and pass into the medulla oblongata beneath the pons, and then pass through the medulla to the posterior external portion, at which point they enter Deiters' nucleus. It is here that the tract divides into two paths and make up the "Y" indicated above. One arm of the "Y" goes through the triangular nucleus entering the posterior longitudinal bundle, which is situated along the posterior aspect of the medulla and pons, and from here connecting with the nuclei of the third, fourth and sixth nerves. The other arm of the "Y" passes from Deiters' nucleus through the inferior cerebellar peduncle to the cerebellar nuclei. The fibres from the vertical canals take a similar course up to the "Y" formation, this taking place not at Deiters' nucleus, but posterior to it and external to the posterior longitudinal bundle. One arm of the "Y" connects with the eye nuclei and the other goes to the cerebellum by way of the middle cerebellar peduncle. It may be stated here that all authorities are not in accord as to the exact pathways by which impulses reach the cerebellum; that is, through which of the peduncles they pass.

We have now to deal with one more set of pathways; namely, those which have to do with the conscious elements of this

phenomena. In the process of producing a nystagmus, the eyes are first drawn in one direction, let us say, horizontally to the right; this is the slow component. The patient then becomes conscious of this altered position of the eyes, sends impulses from the cerebrum back to the eye muscles and in an attempt to compensate for this condition the eyes are quickly drawn in the opposite direction, producing the quick component of the nystagmus. The exact anatomical pathway is not known. Likewise, there is a pathway by which the patient interprets in his field of consciousness the vertigo or dizziness.

With this brief sketch of the anatomical distribution and function of the pathways in mind, and having established what reactions occur in normal individuals, we are now in a position to interpret any departure from the normal. Any interruption of impulses at any point along these pathways will produce abnormal responses in the form of increased or decreased nystagmus, increased or decreased amount of vertigo, failure to past-point, or absence of falling, and so on. We are able still further by bearing in mind the distribution of the pathways to localize in rather narrow limits the area involved, whether it be functional or pathologic. It is with the foregoing in view that we may proceed with the analysis of the cases.

ANALYSIS OF CASES.

The ages of the 35 cases examined range from 12 to 68 years, with half the cases being between the ages of 15 and 35 years. Fifteen males and 20 were females. Twenty-four had attacks classed as grand mal, and 11 as petit mal. The duration of the disease ranged from two years to 45 years, with a frequency ranging from 15 a day to one in three or four months. One case that had been under observation for several years had not had an attack for nearly a year previous to the examination.

Thirty patients had normal hearing; three, mixed deafness; one conductive deafness, and one advanced perceptive deafness. Twenty-eight patients were on luminal and had been for some time before tests were performed, and it is interesting to note that luminal had no depressing effect on the responses from stimulation.

SPONTANEOUS PHENOMENA.

a. All showed normal ocular movements, except one that presented a slight horizontal nystagmus to the left.

b. One presented a suggestive Romberg.

c. Six cases had spontaneous past-pointing defects, and four of the six showed mental defects.

AFTER-TURNING RESPONSES.

a. In all cases the nystagmus was horizontal in the proper direction, and in no case was conjugate deviation noted. Twelve cases showed normal after-turning nystagmus; that is, a duration of nystagmus of 26 seconds. Eighteen were subnormal, ranging from 14 to 20 seconds. Five were hyperactive, with a nystagmus lasting 30 seconds or more.

b. Only two cases showed pallor, sweat, nausea or vomiting, and one of these was a case that had not had an epileptic attack for nearly a year, and might be classed as a normal individul.

c. The after-turning motion-sensing approximated closely the time period of the after-turning nystagmus, and ten showed normal motion-sensing, 23 subnormal, and the two cases with nausea and vomiting showed a motion-sensing lasting longer than 30 seconds.

AFTER-DOUCHING RESPONSES.

a. The time at which the nystagmus came on ranged from 25 seconds to four minutes, 19 cases out of the 35 requiring more than one minute douching and five more than three minutes, the remainder being at or near 40 seconds. The nystagmus in all cases, with the head upright, showed the usual rotary direction to the opposite side, being equal for the two eyes and in no case showing conjugate deviation. With the head back, the nystagmus was horizontal to the opposite side.

b. In the pointing tests, 16 showed normal past-pointing with both hands; 14, subnormal past-pointing with both hands; four, absence of past-pointing with one hand or the other, and one past-pointed in the wrong direction.

c. The findings as regards the constitutional responses were striking, in that 19 showed no constitutional responses at all; that is, no pallor, sweat, nausea or vomiting, which are very constant findings in the normal.

d. In testing the falling after douching, about half the cases had no tendency to fall, while most of the remaining half showed swaying, with a tendency to fall, while six showed a definite falling to the side douched. One case had an epileptic seizure while the tests were being conducted.

COMMENT.

In commenting on the foregoing analysis, it is evident that a large percentage of active idiopathic epileptics show some departure from the responses obtained in normal individuals. There is, however, no one response which is constantly lacking in all the cases. One cannot help being impressed with the large number that show absent or subnormal constitutional responses, in that after this more or less violent ear-stimulation they are not disturbed, showing no pallor, sweat or nausea whatever.

Quite similar responses are encountered in but two other conditions, these being cerebrospinal syphilis and arteriosclerosis of the cerebral vessels. Both these conditions give subnormal responses, but the absence of the constitutional responses are not so marked as in this disease.

Epilepsy is a disease of great antiquity, having attracted the attention of medical men from the days of Hippocrates to the present hour, and while our knowledge concerning its types and clinical manifestations has been enriched, the true nature of this malady, its pathogenesis and mechanisms, are still a long way from a satisfactory solution. Modern investigators in this field no longer consider epilepsy as a clinical entity, but are inclined to speak of it as a symptom-complex or syndrome. The pathogenesis of this disease has been the subject of a great deal of speculation and has not escaped even the most extravagant fancies. The cause has been ascribed to pathological conditions ranging all the way from congenital cerebral anomalies to gastrointestinal autointoxication. There are also observers who believe that the anatomic changes are purely secondary to the seizures.

The phenomena accompanying the aura have long interested the otolaryngologist. There are often auditory perceptions and hallucinations, peculiar unpleasant sensations of taste and smell, as of burning sulphur or burning flesh, and the optic hallucinations of terrifying red colors, sparks and scintillating scotomata. The aura is rapidly followed by the convulsive stage in which the patient almost immediately loses consciousness and falls. The clonic stage of the convulsion then ensues, in which the head is held retracted and rigid, as if in a defensive attitude. It is this stage of the seizure that many modern investigators believe arises in the basal ganglia. This is soon followed by the motor phenomena of rhythmic violent jerks of the extremities, facial muscles and the nystagmus.

CONCLUSIONS.

With a total of only 148 reported cases it seems too small a number to permit of any definite conclusions, even of a tentative nature. However, it is generally accepted that the vestibular pathways go through the brain stem and are intimately associated with the basal ganglia, and that a failure to get a normal response in the epileptic would seem to indicate at least that some of the pathology of this disease is located in the region of the basal ganglia. This is offered only as a suggestive link in the chain of evidence tending to show that epilepsy is not entirely a cortical disease, as once thought, but that at least a phase of the seizure does arise from the lower centers.

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MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION ON OTO-LARYNGOLOGY.

Meeting of October 12, 1934.

The scientific program was as follows: Dr. John J. Hochfilzer reported the following case of Traumatic Aneurysm of the Temporal Artery:

This patient, who is 18 years old, was in an automobile accident a year ago. He was thrown through the windshield and suffered several lacerations in the face and a rather deep laceration at the tragus of the right auricle. He received first aid at Ancker Hospital and was then sent to his private physician.

A few weeks after the accident the patient noticed a peculiar throbbing sensation in front of the right ear, consisting of a beat and then a sizzling sound in this region. At no time had he any pains or discomfort. Gradually these symptoms increased, the sounds becoming more pronounced and rather annoying. He also noticed a slight swelling in front of the right ear.

Physical examination was essentially negative except for the findings in the right temporal zygomatic region. Over the right tragus there was a vertical scar about three-fourths of an inch long and a marked swelling about the size of a walnut was noticeable over the temporal region. On auscultation over this elevated area, one finds a systolic thrill and bruit. By compressing the external carotid artery and its branches, all these symptoms disappear, so it may be assumed that this is a traumatic arteriovenous aneurysm of the right superficial temporal artery and vein.

Generally speaking, a venous arterioaneurysm is a direct communication between an artery and vein; therefore, it would be better to call it an arteriovenous fistula. One may differentiate between a congenital and an acquired arteriovenous aneurysm. The congenital ones are rather rare and usually refractive to treatment. The acquired ones are 90 per cent accidental and are made possible by the common juxtaposition of large arteries and veins in the extremities or neck, where a perforating wound can establish direct communication between the two vessels.

The diagnostic symptoms of an arteriovenous aneurysm are:

1. The vascular bruit, which can be heard best over the communication. This bruit, which may be accompanied by a thrill, is due to the passage of a forceful current of arterial blood through a small opening from an area of high pressure to one of low pressure.

2. The veins, which must carry this arterial pressure without an intervening capillary bed, dilate and form large pulsating anastomosing masses and so cause enlargement of the region involved.

3. If blood is taken from the aneurysm and the oxygen content determined, one finds a much higher oxygen saturation than in normal venous blood, which is around 62 per cent. In arteries it is 95 per cent.

4. Variations in blood pressure.

Prognosis: In untreated arteriovenous aneurysm the prognosis is uncertain. As a result of infection or increasing hematoma, some may lead to serious consequences, to gangrene or death. Most of the cases run a very chronic course, especially when the communication is in the peripheral vessels. Old

arteriovenous aneurysm lesions may rupture and give rise to serious hemorrhage. Cases have been reported, however, where arteriovenous aneurysm has healed spontaneously. Thrombosis is, perhaps, the most common cause for the spontaneous healing. Finally, out of an arteriovenous aneurysm a truly vascular tumor, such as an angiosarcoma or endothelioma, may develop.

Treatment: 1. All non-operative treatments, such as compression or injection of coagulating fluids or electrocoagulation, are unsuccessful.

2. As to the time of operation, one should wait at least six months; first, because in this time a spontaneous healing may occur; and, secondly, a vast collateral circulation has developed in the meantime.

3. Proximal and distal ligation of the arteries only is insufficient because intermediate branches may keep the fistula alive by supplying the vein with arterial blood. Ligations of the veins also fail for the same reason.

Quadruple ligation is widely employed and has yielded very good results, but all branches arising from the fistula must be included in the ligation, otherwise this procedure is also a failure.

Extirpation of the arteriovenous fistula has given by far the best results and should be the procedure of choice, if the location of the fistula permits it. In intracranial arteriovenous aneurysm, extirpation is naturally impossible.

Glancing at the topographical anatomy in the case presented this evening, one finds the temporal artery branches off from the external carotid just above the internal maxillary artery; it is first covered by the parotid gland coming to the surface just in front of the tragus of the auricle, covered only by the skin and the fascia parotide masseterica. A few millimeters further on it divides into the ramus frontalis, parietalis and zygomatic mandibularis. The superficial temporal vein takes a similar course, emptying into the posterior facial vein, which again empties into the internal jugular vein. The superficial temporal artery and superficial temporal vein are in front of the tragus in very close juxtaposition — hardly one millimeter apart; therefore, one can realize very easily that a cut in the vicinity of the tragus perforated both vessels.

The best treatment in the case presented naturally would be extirpation of the fistula, if that is feasible. One must reckon with the close proximity of the parotid gland and the branches of the facial nerve. If extirpation should fail, we must resort to quadruple ligation.

Dr. Gordon B. New, the retiring President, then read his Presidential Address, entitled: "The Curability of Malignant Tumors of the Upper Jaw and Antrum." Numerous lantern slides were shown.

DISCUSSION.

Dr. Wm. T. Peyton (by invitation) said he was very much interested in this subject, having reviewed the records at the University Hospital and published a report on carcinoma of the antrum. Thirteen cases were included in that report, with one case living and well more than five years after treatment. Since that time they had had two or three more who are still free from recurrence. Dr. Peyton thought the percentage of five-year survivals should be at least 25 per cent now. He believed that the treatment of this condition is now pretty well standardized and results are not going to improve very much until something new is developed in the treatment of cancer. He said that Dr. New's method of opening the floor of the antrum for exposure, drainage and destruction of the bulk of the tumor mass, and radiation for destruction of the remaining portion of the carcinoma, was pretty well accepted at the present time.